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XXI.

RECURRENT AND ABDUCTOR PARALYSIS OF THE LARYNX. INTRODUCTORY REMARKS ON ANATOMY AND PHYSIOLOGY. ETIOLOGY OF PARALYSES OF CENTRAL ORIGIN.*

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The subject, selected by your Council for general discussion, is in my opinion the most interesting and at the same time the most difficult one in the whole domain of laryngology. It is interesting, because it leads the laryngologist at once beyond the sphere of his specialty, and it is difficult, as it deals with the most intricate questions of neurology, about which in many instances the investigators entertain conflicting opinions.

For a full understanding of the part assigned to me—the central causes of recurrent and abductor paralyses—a clear conception of the motor centers in the cortex and bulbus, of

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their predominant functions, their connecting strata, is absolutely necessary. With the approval of your presiding officer I shall briefly analyze the anatomic features and add such physiologic and pathologic data, as have a direct bearing on our subject.

I.—REMARKS ON ANATOMY.

The larynx has to perform two functions, diametrically opposite each other, viz., phonation and respiration. The chief center for phonation, adduction of the cords, a voluntary act, is in the cortex; for respiration, abduction of the cords, a vital process, in the bulbus. In addition there are subordinate centers in reverse order and of lesser dignity for phonation in the bulbus, for respiration in the cortex. The connecting nerve fibers run from the cortical to the bulbar center and vice versa, through the internal capsule. The appended diagram from Rethi[1] shows very plainly the different motor centers and their relationship to each other.

The cortical motor center for phonation was discovered by H. Krause[2] to be located at the descending surface of the prefrontal convolution. The result of his investigations was confirmed by Semon and Horsley,[3 4] who by extending their researches found additional motor areas in the cortex for acceleration and for deepening of the respiration. By ingenious experiments, Risien Russell[5] succeeded in finding below the phonatory center a locality from which abduction of the cords can be elicited. The illustration of the different cortical motor centers is a copy from Semon's treatise in Heymann's Handbook of Laryngology and needs no further comment.

The bulbar representation of the larynx is in the fourth ventricle, and we find in the alea cinerea two localities, irritation of which produces typical abduction of the cords, the respiratory act; further, smaller areas for bilateral abduction, one for unilateral abduction and one for the cadaveric position of the cords, all of which are graphically shown in the illustration, which is also taken from the same treatise by Semon.

The existence of the subordinate centers, mentioned above, has been proven by repeated experiments on animals, of which one only will be cited. Acephalic monsters and animals, whose brains have been extirpated, are able to utter a cry, due to the subordinate phonatory center in the bulbus, but their respira-

tory movements are not materially influenced, confirming the bulbus to be the main center for respiration.

These elementary anatomic data, condensed within the narrowest possible compass, have almost universally been accepted; the first real discrepancy of opinions we meet, is about the question of the root of the motor nerve of the larynx, which is not definitely decided at the present day.

After the careful experiments of Schech[6] the laryngeal motor nerve was for many years considered to have its source from the inner branch of the accessory Willisii, till Onodi[7] and Grabower[8 9] brought forth forcible arguments in favor of the pneumogastric, exceptions to which were taken by some authors. But a recognized authority, H. Oppenheim, says (1905) in his book[10]: there is at present no certainty in regard to the participation of the accessory nerve in the innervation of muscles supplied by the pneumogastric.

The illustration from Edinger shows very clearly the intimate relationship of the nerve nuclei, the motor fibers being in black, the sensory fibers in red color.

Another much debated issue relates to the nature of the recurrent nerve. Whilst many investigators strongly believe it to be exclusively a motor nerve, others offer good reasons, based on animal experiments, to consider it a mixed nerve, carrying also centripetal fibers. But if the latter view should prove to be correct, we cannot yet draw any definite conclusions from it in regard to the human system, as even in animals a differentiation exists in the response in different species.

The last and a most important anatomic fact is the discovery by Risien Russell[11] of the existence of special nerve fibers for adduction and abduction, which run in separate bundles throughout the whole course of the recurrent, those for the abductors on the inner, those for the adductors on the outer side of the nerve trunk. They can be dissected to such an extent, that one set of fibers responds to electrical irritation exclusively by adduction, the other exclusively by abduction of the vocal cords. By his work the formerly vexed question was solved, how a supposedly homogeneous nerve could effect such directly antagonistic movements.

II.—REMARKS ON PHYSIOLOGY AND PATHOLOGY.

The cortical motor centers of the larynx differ from other cortical motor centers in the essential point that irritation of either one is always followed by symmetrical bilateral adduction of the cords, phonetic movements, which also take place when one cortical center has been destroyed by disease or experimentally removed. This physiologic observation had enjoyed universal acceptance, till Masini[12] maintained that he found a locality in the cortex, from which movements of the opposite side of the larynx could be produced. Subsequent experiments by others did not verify his statements, but recently Katzenbach[13] obtained by an improved method similar movements of the corresponding as well as opposite side.

The influence of the bulbar center on respiration and on the position of the glottis has been carefully studied by Semon[14] in his endeavor to ascertain if the vocal cords play an active part during quiet respiration, and to determine the cause of the difference between the width of the glottis during life and its cadaveric position after death or after severance of the pneumogastric or recurrent. He found by numerous and carefully made measurements with graduated mirrors, that in over 80 per cent of adults the glottis remains stationary during quiet respiration and is considerably wider than in the cadaveric position. Its average width is in men during life 13.5 mm.; after death 5 mm.; in women 11.5 and 4 mm. respectively. Without entering into a detailed statement of his experiments and arguments, he is led to the conclusion, that the greater width of the glottis during life is the result of a permanent activity of the abductor muscles, due to tonic, rhythmic impulses, received by their ganglia centers in the medulla from the neighboring respiratory center through centripetal fibers of the pneumogastric. He calls this physiologic action of the abductor muscles the abductor tonus.

I cannot conclude my introductory remarks without calling to your attention and emphasizing the far-reaching importance of the proclivity of the abductor muscles to paralysis in progressive organic lesions.

Whilst Rosenbach,[15] on the strength of a case of cancer of the esophagus, accompanied in the beginning by paralysis of both abductors, later of both recurrent nerves, was the first

to proclaim that the abductors are the first to suffer from compression of the recurrent, the credit belongs to Semon of proving that this important pathologic condition is seen not only in local lesions, affecting the nerve along its course, but also in central cerebral as well as spinal diseases of an organic character.

His first publication[16] appeared (1881) in an American journal of high standard, devoted to our specialty, but meeting with a premature death on account of the indifference of the laryngologists at that time, viz., the *Archives of Laryngology*. His statements met with a host of opposition and elicited controversies, sometimes of an acrimonious character and not always of the highest scientific standard. By his incessant labors extending from 1881 to 1897 and by publications too numerous to quote, he had finally the satisfaction of having the so-called Semon law universally acknowledged.

It is beyond the scope of these remarks to enumerate the arguments he put forth to support his assertion, the truth of which all of us have had undoubtedly frequent occasions to confirm.

The ultimate, true cause of the proclivity of the abductors to paralysis has not been definitely determined. Grabower[17] sought not long ago to solve the problem by anatomic, instead of experimental research, previously employed. He examined the innervation of the laryngeal muscles in regard to the configuration of their last nerve terminals, and counted the number of nerve fibers in each muscle. He found, that the branch for the abductor does not divide into smaller ones, radiating in different directions, as in the adductors, and that the number of nerve fibers is less in the former than in the latter. The posticus has on the average 281 terminal fibers, the external and internal thyroarytenoid 485, the transversus 279, and the sum of all the adductor fibers is 680.

I have so far not been able to find reports confirming Grabower's investigation, but without feeling competent to pass on its merits, I am free to say that I would consider it a most acceptable solution of the question at issue. It would explain also in a natural way by an anatomic anomaly the first and only exception to Semon's statement during the long period of twenty-five years, observed by Saundby[18] in a case of esoph-

ageal cancer, the analysis of which I have to leave to the gentleman treating of peripheral causes.

Semon[19] admits frankly the facts in Saundby's case, but is not able to offer an explanation. On the occasion of his visit to the United States in 1904, I called his attention to Grabower's publication, but the same seemed not to appeal to him. He adheres to his belief in the biologic differences in the structure of the laryngeal muscles and nerves, which is evident from experimentation in young and old animals as well as in different species, and still more from the differential influence of superficial and deep narcosis on the recurrent, which our late member, Hooper,[20] was the first to investigate.

III.—ETIOLOGY.

Recurrent and abductor paralyses of central origin are caused by organic affections, whilst cortical paralysis of a functional character occurs in hysteria, psychic emotions, shock, etc., with concomitant paralysis of the adductors, but not of the recurrent proper.

According to the location and nature of the diseases we can distinguish paralyses due (1) to cortical lesions, (2) to disturbances of the nerve tract in the internal capsule between cortex and bulbus, (3) to bulbar processes, (4) to syphilis, (5) to tumors, exostoses at the cranial base, pressing upon the pneumogastric, (6) to spinal diseases, (7) final paralyses of the Avellis and Hughling Jackson type.

Beginning with paralyses due to cortical lesions, we enter at once a much contested field.

A number of cases have been published in which the authors claim that an unilateral cortical affection had caused an unilateral organic paralysis. Whilst in many instances the history, the laryngeal examination, the postmortem reports, are incomplete or missing, and therefore the proof of the contention made is lacking, there are cases in which the symptoms in the living corresponded with the lesion found after death. Garel,[21] for instance, saw a woman 73 years of age with hemiplegia of the right side and complete paralysis of the left vocal cord. The autopsy showed softening of the left hemisphere

and two similar foci in the right, one at the base of the third frontal convolution, to the latter of which Garel attributed the laryngeal paralysis. A microscopic examination of the bulbus had not been made, and consequently its integrity cannot be vouched for.

On the other hand, cases which appeared during life to belong to this class, showed bulbar disease after death. Our member Delavan is quoted amongst the above authors, when relating the history of his patient seen during life,[22] but in his comment on the result of autopsy, he says[23] that his case was a direct proof that paralysis of the larynx of central origin is generally due to bulbar lesion and not to disease of the cortex.

In my opinion there are at the present time extremely few believers in an unilateral laryngeal paralysis brought about by unilateral cerebral disease; otherwise the number of cases would certainly be much larger than we find in the literature, as unilateral cortical lesions, for instance hemorrhage, softening, etc., are not infrequent. Semon in his article in Heymann's Handbook, 1898, could collect only twenty-five cases in which such a coincidence was claimed by the authors. The strongest argument against the occurrence of these paralyses is the anatomic fact that laryngeal movements can be excited by each of the two cortical centers, independent of the loss or destruction of one of them; further, that the experimental extirpation of the cerebral hemispheres, down to the fourth ventricle, does not interfere with the respiratory movements of the cords, the integral function of the abductor.

Affections involving both cortical centers, for instance hemorrhages, are as a rule, not confined to this area alone, and are of such a serious character, that the laryngeal condition is overshadowed by the grave general symptoms. But cases are reported, in which the connection between the cortical and bulbar center through the internal capsule became obliterated by the morbid process with resulting aphasia.

Eisenlohr's[24] patient had general motor disturbances, was absolutely speechless, and the postmorten showed destruction of the posterior third of both thalami optici, partial degeneration of the posterior part of the internal capsule and of the pyramidal tract of the medulla and spinal cord. Pathologic

changes of a similar nature were found after death and reported by Pitt[25 26] in a case of chronic nephritis with two apoplectic attacks in quick succession, the second one being followed by loss of voice. In both cases bulbar and cortical lesions were absent and the patients experienced no respiratory difficulties during life.

Similar laryngeal symptoms, associated with lingual and labial pareses are observed in pseudobulbar paralysis, due to disseminated cerebral lesions, in which disease of the medulla and respiratory troubles are absent, and the laryngeal muscles undergo no fatty degeneration.

The subsequent chapters can be considered more briefly, as they deal mostly with well known data, and offer no field for widely dissenting views. Under bulbar paralyses I have included all cerebral diseases, irrespective of the primary seat being in the bulbus or in the hemisphere. Further, the etiologic features of recurrent and abductor paralysis will not be treated of separately, as both are liable to be caused by the same affection, and as the latter is in many instances only a precursor of the former. Prevalence of one or the other type in special diseases will be mentioned in its proper place.

Progressive bulbar paralysis, being due to a degeneration of the motor nuclei in the medulla, represents a genuine type of the bulbar origin of laryngeal paralysis. As a rule paralysis of other organs, tongue, lips, precedes the laryngeal symptoms, which appear sooner or later, dependent on how early the laryngeal nuclei are attacked. The laryngoscopic picture varies in different patients, according to the focus and the intensity of the process. Complete recurrent and unilateral as well as bilateral abductor paralysis has been observed, whilst some authors claim to have seen also adductor paralysis.

Another bulbar affection occupying the same or also adjacent areas, is the apoplectic form of bulbar paralysis, caused by disease of the blood vessels of this region, producing hemorrhages, thrombosis with subsequent softening, also embolism of the vertebral or basilar arteries. Many reported cases of cerebral hemorrhages and encephalomalacia with concomitant laryngeal disturbances are undoubtedly due to such lesions, which have been described already as early

as 1879 by Eisenlohr.[27 28] If the patient survives the attack and its sequelae, we can expect to find a recurrent paralysis, if all the motor fibers are destroyed, otherwise an abductor paralysis.

In the different forms of meningitis we observe anomalies of sensation, but seldom paralysis of the larynx. Our late member Major,[29] saw a patient with bilateral abductor paralysis after cerebro-spinal meningitis; tracheotomy was performed and the patient's condition remained stationary for seven years, when he died from an accident.

Cases of paralysis from multiple cerebro-spinal sclerosis are rare, and the laryngeal symptoms depend on the extent of the disease. Loeri,[30] reported several cases, one of which had recurrent paralysis, J. Solis-Cohen[31] (1886) a case of unilateral, later bilateral abductor paralysis, shown to this association seven years before. From the carefully made autopsy he concludes, that the paralysis has been caused by an ascending sclerosis. Rethi[32] collected in a recent publication 42 cases of sclerosis from the literature and gave a full description of the subject in all its bearings.

Although infectious and toxic paralyses do not belong to the part assigned to me, I cannot refrain from mentioning syphilis of the brain as a cause of recurrent or abductor paralysis. M. Mackenzie[33] published already in 1873 one case, and Lefferts,[34] 1878, two cases of bilateral abductor paralysis due to syphilis, all three cured by specific treatment.

Tumors at the base of the brain are liable to produce paralysis by pressure on the pneumogastric. The symptoms are generally not confined to the larynx alone, as the extension of the growth is apt to implicate other nerve roots also. Nothnagel[35] communicated an exceedingly interesting case of an extended unilateral paraylsis, involving the recurrent of the same side, which was due to pressure from a large abscess at the cranial base, found at the autopsy. Similar symptoms we must be prepared to meet in affections of the cranial bones, exostoses, fractures of the base of the skull, etc.

The importance and the deleterious influence of locomotor ataxia on the larynx is too well known to all of us to need but brief remarks. General medicine is indebted to laryngology because in more than one instance the laryngologist detected

abductor paralysis in an otherwise healthy person with a perfectly clear voice, and suspicion being aroused, the examination of the patient revealed locomotor ataxia. By far the most prevalent type is abductor paralysis, of which again the bilateral form is more frequent than in any other disease. Burger[36] collected in his excellent monograph 71 cases of paralysis in tabetics, covering the period from 1866 to 1891, forty-three of which had abductor paralysis. Still more striking is Sendziak's[37] supplementary table of 53 cases of paralysis published between 1892 and 1898. Of these six only had recurrent, of the balance 22 unilateral and 25 bilateral abductor paralysis.

In the remaining spinal diseases we find only isolated cases of laryngeal paralysis, a few of which may be mentioned. Loeri[38] observed a case of bilateral recurrent paralysis in amyotrophic lateral sclerosis, and one in acute spinal leptomeningitis, Schroetter[39] and Gavello,[40] each a case of unilateral recurrent paralysis in syringomyelia, and Koschlakoff,[41] a bilateral abductor paralysis in progressive muscular atrophy.

Although finding scant consideration in the text books, a series of unilateral laryngeal paralyses, associated with such of other organs is of sufficient interest to be mentioned.

Avellis[42] was the first to draw attention to the occurrence of simultaneous unilateral paralysis of the larynx and soft palate—the genuine Avellis-syndrome. In another form the muscles of the neck are also paralyzed. If hemiplegia of the larynx, pharynx and tongue exists, we have the Hughling-Jackson type. A typical case of the latter with abductor paralysis was demonstrated in the London laryngological society by Semon,[43] who in consideration of the general symptoms, believed it to be of central origin, due to a meningeal thickening at the base of the brain. Tapia,[44] who observed unilateral paralysis of the larynx and tongue, called it the fourth type, and finally Pohli[45] gave a complete description of the subject in the *Italian Archives,* 1906.

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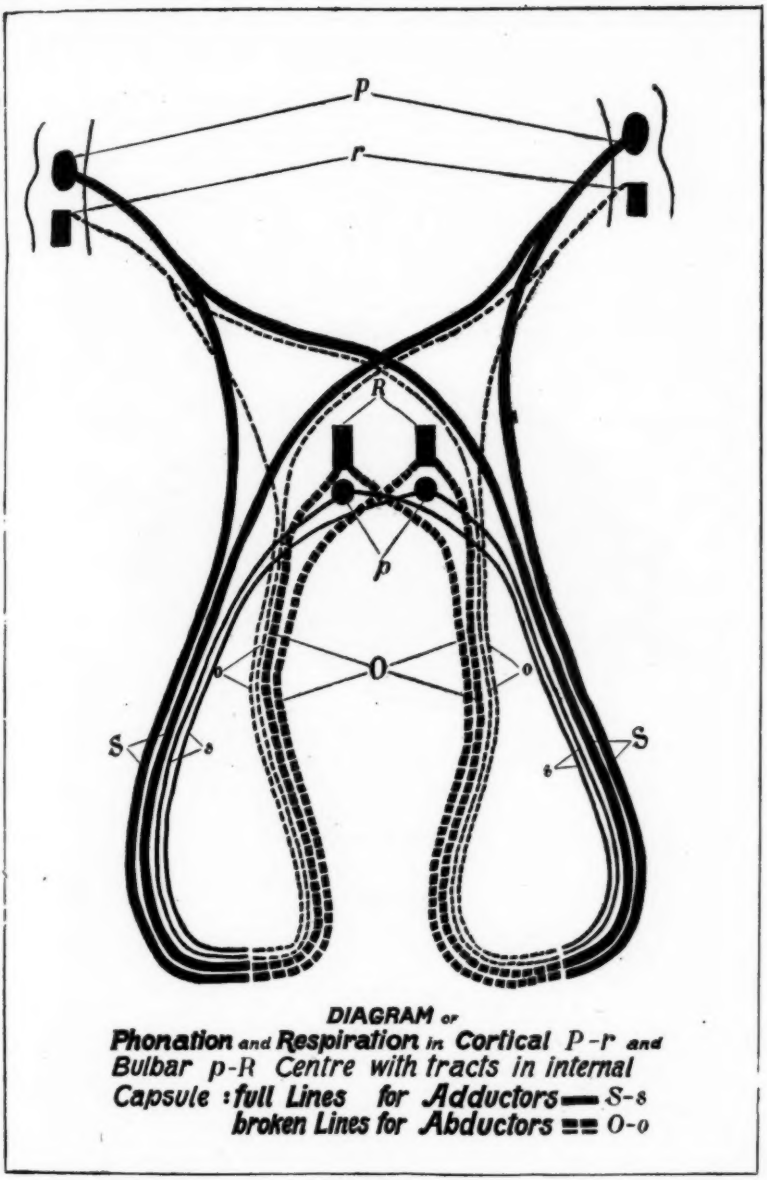

DIAGRAM of
Phonation and **Respiration** in **Cortical** *P-r* and
Bulbar p-R Centre with tracts in internal
Capsule : **full Lines for Adductors** — *S-s*
broken Lines for Abductors == *O-o*

FIGURE 1.

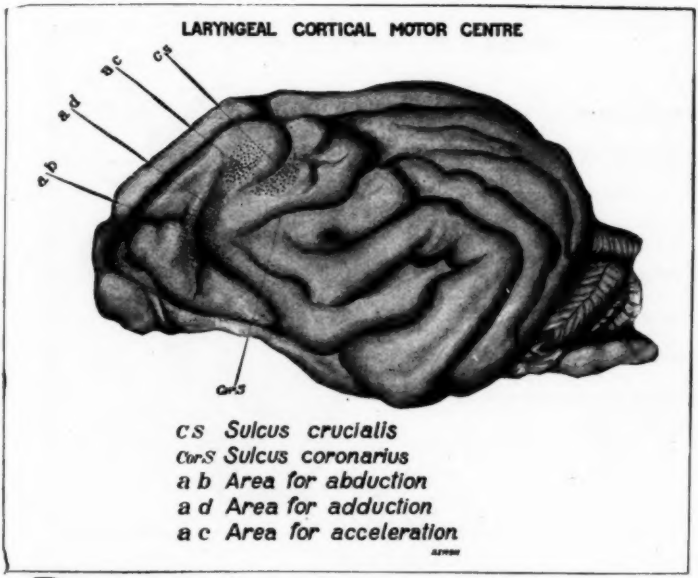
LARYNGEAL CORTICAL MOTOR CENTRE
a c
a d
a d
a b
CorS
cs Sulcus crucialis
CorS Sulcus coronarius
a b Area for abduction
a d Area for adduction
a c Area for acceleration

FIGURE 2.

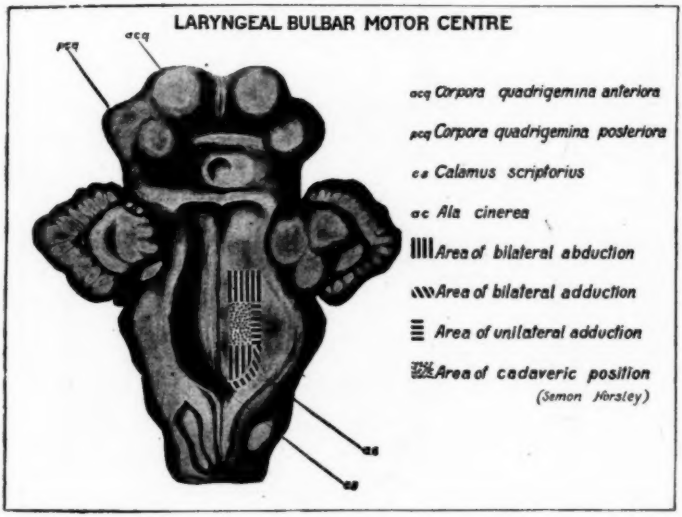
LARYNGEAL BULBAR MOTOR CENTRE
pcq
acq
acq Corpora quadrigemina anteriora
pcq Corpora quadrigemina posteriora
cs Calamus scriptorius
ac Ala cinerea
Area of bilateral abduction
Area of bilateral adduction
Area of unilateral adduction
Area of cadaveric position
(Semon Horsley)

FIGURE 3.

NUCLEI OF CEREBRAL NERVES

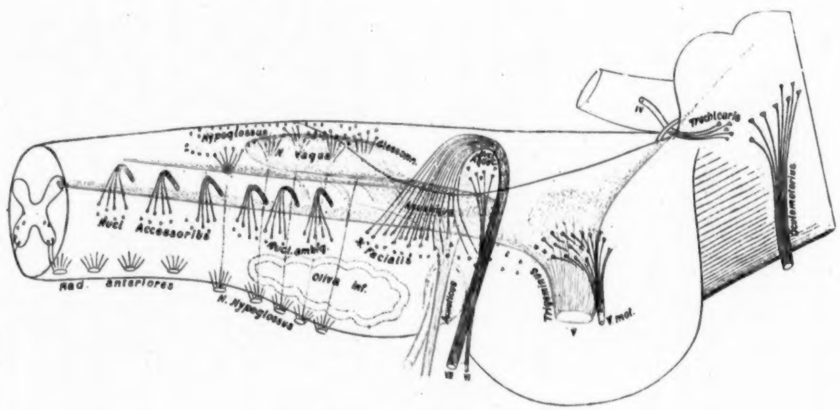

FIGURE 4.

XXII.

RECURRENT AND ABDUCTOR PARALYSIS OF THE LARYNX. DIAGNOSIS AND TREATMENT.*

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In the diagnosis of unilateral recurrent nerve paralysis, which when complete results in the fixation of one vocal cord in the cadaveric position, the cracked, softly raucous voice, is suggestive rather than distinctive, but it is usually the one small symptom of an inversely grave condition which first causes the patient to seek advice. It is nature's distress signal, full of meaning to him who reads the code. The same is true of that partial stridor which may or may not usher in an unilateral abductor paralysis in which the dilator muscle alone being powerless, the vocal cord is retained in a position closing over one-half of the lumen of the larynx. Either symptom demands a prompt resort to laryngoscopic inspection, but even then by reason of the fact that patients are liable to present themselves for the first time during "a cold" or other aggravating influence, confusing departures from the typical image may be encountered, and one turns next to the really vital phase of the diagnosis; a search of the chest, neck and central nervous system, for the cause and significance of the vocal paralysis. The tortuous course and complex composition of the recurrent nerves should be kept well in mind. Branching from the pneumogastric, the left one takes a turn under the arch of the aorta, *between it and the left bronchus,* upward to the larynx, while the one on the right passes under the subclavian artery; both being liable to compression by aneurysm, mediastinal and cervical tumors and, on the right, by

*Read as part of a Symposium before the American Laryngological Association, May 11, 1908.

disease at the apex of the lung or its pleura, which is crossed by the nerve. The similarity of lettering of the two words abduction and adduction is unfortunate, for in addition to typographic errors, a conscious mental effort is necessary to avoid confusing one with the other, but as applied to the movements of the vocal cords, they are more exact in meaning than any other available terms. The opposing muscles which effect the to and fro motion of the vocal cords, adduction for vocal vibration, abduction for breathing space, are innervated by different fibers, which merely travel in the same recurrent nerve trunk, either set of fibers alone to any degree or both sets together, being subject to paralysis. Semon's law refers to the fact that the abductor fibers and muscles are the more vulnerable of the two, yielding first to an equal pressure on the trunk, and in consequence every complete paralysis of a vocal cord by pressure must have passed through a preliminary stage, however brief, of abductor paralysis alone.

ANEURYSM GROUP.

The typical image of complete unilateral recurrent nerve paralysis illustrated in Fig. 1, is introduced as a standard from which to describe the diagnostic variations. It represents the larynx of a middle-aged salesman, whose voice, he said, "Failed all at once, one day, while selling goods." Not only is the left vocal cord motionless in the cadaveric position, but it is relaxed, tends to bay outward, and its arytenoid, which is *not swollen,* has "fallen in," i. e., collapsed forward, which shortens the vocal cord and permits the cord to sink to a plane slightly below the level of its fellow. On phonation, the left arytenoid may give a twitch, but fails to straighten up or convey more than a mere tremor to the cord, while the opposite arytenoid and vocal cord, in an effort to compensate, overreach the center line, the sound arytenoid crossing a trifle usually in front or possibly behind the paralytic one and the healthy cord coming nearly but not quite over to the paralyzed one. These are not insignificant details for their presence collectively, rather than the cadaveric position alone of the cord, insures the fact of a complete recurrent nerve paralysis. Its cause in this instance was equally evident. There was substernal pain and a radial pulsus differens, but no other symptom, excepting that in the fluoroscope, a small pulsating

aneurysm of the arch of the aorta was visible. Five months later, while walking on the street, this patient fell dead with a gush of blood from the mouth—the aneurysm had burst; presumably into the left bronchus.

Life's duration in aneurysm, however, is seldom so brief. Dating from the early period of vocal paralysis, it has often surpassed my expectation; in one 10 years, in another 5, while others are now living at 2, 3 and 4½ years. The seemingly prolonged course, last mentioned, the patient being still active, is explained by the fact that it dates from the earliest demonstrable stage in the development of the aneurysm, as evidenced by the presence of unilateral abductor paralysis, which is the first definite effect of pressure on the recurrent laryngeal nerve. A sketch of the larynx made at that time (Fig. 2), represents the typical image of unilateral abductor paralysis and is intended to show not only the vocal cord motionless in the median or closed position, but also that the arytenoid has not yet "fallen in," hence the cord is but little depressed or shortened and not very much relaxed. Paralysis limited to abduction is not the only possible interpretation of the median position of a vocal cord, for one of the most deceptive of the variations from the typical image of complete recurrent paralysis is a fixation of the cord, not exactly in, but quite near to the median line instead of in the usual cadaveric position. Either form of paralysis is suggestive of an aneurysm, but with paralysis of the abductor fibers alone, indicating an earlier stage and more limited development of the aneurysm, a longer course is to be expected and an "arrest" or even a retrogression might not be impossible. Hence, in confirmation of the memorandum and sketch made of abductor paralysis in this case I will quote the additional notation inscribed that, "The vocal cord was not much relaxed and that the arytenoid, although standing a little forward, had not yet 'fallen in,'" a combination of conditions which I interpret to indicate an incomplete degree of paralysis of the recurrent nerve which, in connection with the median position of the cord, could be no other than an abductor paralysis. His x-ray print I find somewhat blurred by the effects of time, still it distinctly shows, representing the aortic arch, a broadened shadow which rises in the back above the normal limit of the fourth rib, with a bulge to the left which centers near the second rib, and a smaller bulge to the right at the fourth

intercostal space. A fluoroscopic examination also, by Dr. John Kales and the author, was recorded at that time as follows: "A distinct but small pulsating bulge in the descending part of the arch of the aorta could be plainly seen."

Since writing the above description with its deductions relative to the 4½ years' duration of an aortic aneurysm, this patient, known to have remained vigorous so long as to suggest a shade of doubt in the diagnosis, has returned, thus enabling me to complete the evidence to date with respect both to the laryngeal paralysis of which he is conscious only when further disabled by a "cold" as at present, and to the aneurysm which does not even yet cause any subjective symptoms other than the vocal cord paralysis. Re-examination confirms all of the original findings, with natural progression. The position of the left vocal cord has advanced from the typical abductor or first position, partly but not wholly, into that of complete recurrent paralysis. It now exemplifies that variation from the typical image of complete recurrent paralysis described as "a fixation of the cord not exactly in, but quite near to the median line instead of in the usual cadaveric position." Intensified tubular breathing, the physical sign emphasized as coming next after vocal paralysis, is now heard in the back at the left, above the angle of the scapula and, as might be expected with compression of a bronchial tube, the vocal resonance and fremitus are diminished at the same point. A blurred, widely transmitted first sound stands for the aneurysmal bruit and the subclavian impulse is in excess of the natural. The fluoroscope and x-ray plate show a clearly defined sacculated aneurysm with a diffused distension of the rest of the aortic arch.

It requires but a slight expansion of the aorta at a given spot to compress the left recurrent nerve, hence the frequency of even a complete recurrent paralysis at comparatively an early period, when perchance one may search in vain for any other classical sign of the aneurysm, which in fact can still be so small that even a radiograph will leave a doubt behind, as in Fig. 3. Here there was neither bruit, thrill, tracheal tug nor protrusion, yet there was paralysis of the left vocal cord and there was, moreover, a noteworthy accessory physical sign, i. e., an exaggerated bronchial breathing transmitted to the back, a symptom which coming early, pari passu with vocal paralysis, should be of special disagnostic value to the laryn-

gologist, for an aneurysm which compresses this nerve must at its very next step, compress the left bronchus. An appreciable diminution in vesicular expansion comes only later.

It has been noted that in the typical image of complete paralysis, the "fallen in" arytenoid is not swollen, but exceptionally, as a departure from the typical image, a coexisting swelling of the arytenoid is found which is liable to disguise the paralytic aspect and to cause the condition to simulate a crico-arytenoid ankylosis with fixation of the cord. Fig. 4 represents this important variation. When first observed, the right cord was stationary, but in a doubtful position, the arytenoid and subglottic tissue was swollen and the left cord, also, was sluggish in movement. It was only after some weeks of detergent local treatment that the cadaveric position of the right cord could be accurately determined, the left cord, meanwhile, having definitely weakened into the first position, that of abductor paralysis, which in conjunction with the other lesions sufficed to cause an alarming respiratory stridor on exertion or on "taking cold." Aneurysm was now evidenced by other signs and its existence was later verified in Berlin by Dr. Krause, who, in explanation of the swollen arytenoid, wrote: "The small tumefaction of the arytenoid is a little bit unclear, but may be caused by a prolapsus of the cartilage." Hooper,[1] also, once drew our attention to certain "anomalous muscular fasiculi,[2] innervated from other sources, which when present in conjunction with recurrent paralysis would serve to pull the arytenoid forcibly forward." The possibility thus, of a partial dislocation of the arytenoid amounting to a prolapse instead of merely a collapse, is a striking point in diagnosis to be remembered, especially with reference to the differentiation of paralysis from ankylosis, which is rendered thereby all the more difficult.

The bilateral inflammatory form of paresis of the internal tensors with its familiar ellipse, is in no danger of being mistaken for recurrent nerve paralysis, but I have twice observed in clergymen an unilateral internal and external tensor paralysis, induced by vocal strain, but of so severe a grade as to simulate and perhaps in a way to partake of a recurrent nerve paralysis. Assuming for it a neuromuscular rather than a strictly myopathic basis, it is the nearest approach to a direct impairment of the nerve endings which my series affords. The level of the cord is lowered, its range of movement to

and fro lessened, and the arytenoid disposed to collapse, but it is distinguishable by a predominance of the flaccid tensor element, absence of lead, alcohol or other toxemias, absence of any cause of pressure, and it finally recovers by rest to the voice.

Out of a total of forty cases upon which this study is based, the left recurrent nerve was paralyzed by an aneurysm in eight and the right recurrent in six, including in both numbers those in whom the opposite cord was likewise but less affected. Concerning the treatment, in only a few of the aneurysm group was local medication indicated and then not for the paralysis, itself, but rather to aid in strengthening the voice or in diminishing the stridor by controlling the inflammatory complications. Moderation in voice use is enjoined on all and soon has its reward in distinctly improved tones, the voice, in time, through compensation, often becoming fairly good for conversational use. The prognosis of aneurysm, in general, is not so absolutely bad, and discovered at an early stage it should be better. Syphilitic arteritis in other situations, for example, in the brain, elicits and yields to a vigorous specific treatment; why not so in an early stage of aortic arteritis?

PLEURITIS GROUP.

Four additional paralyses of the right cord compose the pleuritis group. All were tuberculous and in none could any other cause be assigned than an assumed circumscribed pleuritis. In one of them, whatever might have been the exact cause of a recent and complete unilateral right recurrent paralysis, which showed a typical image, a perfect restoration of power to the cord, was gradually attained contemporaneously with a treatment by fresh air and rest for the incipient tuberculosis. Our means of certifying a diagnosis in this group are as yet sparse. The x-ray should be of service for the purpose of excluding or establishing the presence of enlarged mediastinal lymphatic glands on the left side, which in tuberculous subjects constitute a possible cause of pressure on the recurrent nerves; but on the right side, enlarged glands so located would scarcely be beyond the reach of deep palpation. However, a radiograph was taken of this particular patient, who recovered, and no abnormality was shown. Her reaction to the tuberculin test was positive.

TUMOR GROUP.

Another departure from the typical image of complete paralysis is presented in Fig. 5, in that the ventricular band is hypertrophied and almost covers over the paralyzed cord, nature having gradually put forward this false cord in compensation; it vibrates with the sound cord and produces a fair voice. It is selected from my next group of eight cases, in each of which a tumor was the cause, and in all but two of which, unlike in the aneurysm group, the vocal disability appeared only as a late and gradual development. In this patient a primary malignant growth had been removed from the chest wall three years previously, a fact which he considered so irrelevant to vocal trouble that he deliberately falsified when I asked about the scar, saying he had been struck by a shell during the war. A grimly humorous episode served, in the interests of science, to complete my record of this case, for the autopsy having been held in my absence, the widow, who it appears had fervently disapproved of his falsehood about the scar, entered one day and dramatically deposited a large package before me on the desk, stating exultingly, "There, that's what he had." It proved to be a sarcomatous tumor from the mediastinum.

In this group also I find another observation, that late in the course of a carcinoma of the esophagus, an irritable cough and weak voice having appeared within a fortnight, the left cord was found motionless, near to, but not exactly at the line of abductor paralysis and that the arytenoid had not "fallen in." Evidently the paralysis was progressing gradually and a remnant of innervation remained.

Esophagoscopy has been helpful, although usually, by the time a vocal paralysis supervenes in carcinoma of the esophagus, the diagnosis is plain. I was once temporarily misled with respect to the larynx by a condition which suggested a dawning vocal paralysis in consequence of esophageal neoplasm. There was post-prandial eructation of what the patient called "a lot of phlegm," but which in reality was a fermented mixture of mucus and food which irritated the arytenoids and enfeebled the voice, but obscured the image. The x-ray disclosed a bulge, and showed the arrested bougie within it (Fig. 6). The esophagoscope, passed under general anesthesia, was arrested at this same point where an easily bleeding mam-

millated mucosa was plainly seen, the exact nature of which, however, it was impossible to interpret. As a palliative treatment in this particular case, which proved not to be one of paralysis, the expedient of assuming an inverted recumbent position after each meal, which by gravity emptied the pouch, has afforded a gratifying relief to the throat symptoms, which two years ago were quite distressing. Meanwhile, the obstruction in the esophagus, whatever its nature, remains still in statu quo. Having the appearance of a neoplasm, it is of interest in this connection because it is differentiated from aneurysm by the fact that it does not pulsate as observed in the fluoroscope, and because it is in line with the dictum that vocal parlysis, unlike in aneurysm, is not an early symptom of neoplasm of the esophagus unless the latter is located high up.

We recognize in the so-called "lazy cord" a symptom of carcinoma of the larynx, but I think it is little known that total immobility of a cord in the cadaveric position may be the first visible evidence of carcinoma of the larynx. Through a surprise of this sort at autopsy, I am able to duplicate the single observation of it which Semon[8] records in these words: "Einmal habe ich eine bösartige Kehlkopfneubildung in Form der Unbeweglichkeit einer Stimmlippe in Cadaverstellung beginnen sehen."

The other exception to a late development in the tumor group occurred with a high carcinoma of the esophagus and involved both cords, the left a cadaveric, the right an abductor paralysis. The end was then approaching, but aphonia two years before had been the very first symptom, the reason being that high up in the neck the recurrent nerves lie so close to the esophagus that not only one but both nerves are liable to be affected by pressure or to be incorporated in the carcinoma almost at the start.

Goitre was responsible but once for a complete paralysis of a vocal cord. It was a small, firm enlargement of the left lobe, which was treated by surgical removal, but the nerve had already atrophied. In goitre, when the voice begins to weaken, the cords should receive the closest scrutiny, for surely the first definite evidence of paralysis would be an additional incentive to surgical interference. This principle pertains equally to other cervical tumors and to enlarged lymphatic glands. Its value was brilliantly evidenced in one of this

group by a complete restoration of voice which followed the excision of three tuberculous glands in a school teacher whose left vocal cord previously had been motionless in the cadaveric position.

The large variety of conditions capable of inducing recurrent nerve paralysis, is emphasized by an error in diagnosis of which I became apprised only when tracing the patients for the purpose of this report. The image of complete paralysis of the left cord was typical and without any tumefaction inside the throat. A swelling in the neck at the site of the thyroid gland was naturally mistaken for a goitre, the subject being a girl of 18 years. Goitre was accepted as the cause of the paralysis and its surgical removal recommended, but not done. Signs of tuberculosis at the right pulmonary apex seemed to explain her anaemic or cachectic appearance. The supposed goitre turned out to be a malignant neoplasm, which terminated in the usual manner about a year later.

CENTRIC BULBAR ABDUCTOR GROUP.

Like other respiratory muscles, the abductors of the vocal cords (dilators) being impelled by reflex action, have their main centers in the medulla oblongata (bulbus), so near together that naturally the symmetrical double abductor paralyses predominate in my bulbar group. The mere fact of symmetry in persistent abductor paralysis is indicative of a bulbar lesion. When uncomplicated the appearance in the mirror is pathognomonic. The flaccid cords which will not separate, their edges flapping up and down like a sail in the wind, are unmistakable. The question of a possible overpowering spasm of the opposing adductor muscles did not need consideration in any one of this class, although it may arise especially in connection with the intermittent stridor of an incipient stage of abductor paralysis and also in the unilateral type. The differentiation from ankylosis may require the same degree of critical observation as recurrent nerve paralysis. As a paralytic immobility is liable to lead into a sort of ankylosis or stiffness of the joints, and moreover there is at least one form of arytenoid arthritis, previously described by me under the name of arthritis deformans of the larynx,[4] which causes a *symmetrical* ankylosis without much tumefaction.

An energetic course of mercurial and iodid medication is

indicated whenever the centric degeneration is connected with tabes or with any other suspicion, however remote, of syphilis, for exceptionally one finds a practical recovery recorded. Eight cases of abductor paralysis compose this bulbar group, four of them being of the persistent symmetrical double type. Unfortunately, in all of these four the varied treatment did naught but eventuate in tracheotomy which, although far from an ideal remedy, remains still the best life-saving device. For eight years one of them had been wearing a tracheal tube of the sort which is fitted with a ball and socket valve which enabled him not only to inspire with freedom, but to talk with ease, for the obstruction by abductor paralysis is inspiratory only, the downward air pressure forcing the vocal cords even closer together, while the outward air pressure balloons them slightly apart. Thus, with the tube in place, by a skillfully modulated use of the expiratory current which served momentarily to close the tubal valve, second to divert the air from the tube through the chink of his glottis, he could uninterruptedly intone and articulate sentences of considerable length. Neither this patient nor another, who had been dependent for his life, during several years, upon a simple tube which in order to effect conversation required its stoppage by the usual conspicuous method of holding the finger at its opening in the neck, would, on account of the risk to speech involved, consider any effort to widen the glottis to the small extent which would be adequate for ordinary breathing, either by means of an excision of one cord or by the proposed method of deliberately causing it to assume the cadaveric position by intentionally cutting the recurrent nerve. The latter method, supplemented perhaps by mobilization of the cryco-arytenoid joints if found stiffened, would seem worthy of a trial, considering that prolonged dependence upon the wearing of a tracheal tube is in itself a menace to life. Neither of them suffered from tabes, but both gave a history of syphilis.

Not only the parasyphilitic degenerations, but also the degeneration incidental to advanced age may happen to strike these minute centers in the medulla. It was the only apparent lesion, without other cause, in a woman of 75 years, in whom a treatment by emollients and mild vascular astringents sufficed, under careful observation, to defer for some months, the ultimate tracheotomy. The same line of treatment in con-

junction with antisyphilitic remedies was finally successful in another one of this group, but of a different type, the condition being a partial and unsymmetrical abductor paralysis in which the stridor became serious only during periods of acute exacerbation by "a cold."

The remaining representation in the bulbar group is of that combination which anatomically is far from clear, now that the spinal accessory nerve is not considered contributory to the recurrent nerve, in that the abductor paralysis being unilateral and devoid of symptoms was discovered incidentally, as the complaints had reference to difficulty in swallowing and articulating, in consequence of paralysis of the palatal, pharyngeal and lingual muscles together with atrophy, in one instance, of the trapezius and sternocleidomastoid muscles. One, the victim of a generalized arteriosclerosis without syphilis, was not responsive to any form of treatment and succumbed from inanition, while another, a syphilitic, was benefited by energetic specific medication to the extent of regaining an ability to swallow liquids without a nasal reflux and to articulate with fair distinctness.

CENTRIC CORTICAL GROUP.

Owing to the bilateral laryngeal action which ensues from an unilateral cortical stimulus, it is supposed that paralysis of one vocal cord from a lesion of the opposite side of the brain, really does not occur and that some fallacy has underlain the few apparent cases which have been recorded.[5] Delavan[6] reports in convincing detail one case previously published as cortical, in which at autopsy was found a conjoined bulbar disease; but probably the fallacy also lies in mistaking the "stroke" of a syphilitic endarteritis for that of true hemorrhagic apoplexy. Laryngeal paresis can, of course, ensue from organic cortical lesions which affect the brain centers of both sides or interrupt the nerve fibers in transit from both sides, as in syphilitic degenerations or arteriosclerosis; and the six cases which compose my centric cortical group may have been either of this nature or else similar to Delavan's case. In three of them, the vocal paralysis occurred in connection with attacks of so-called apoplexy, but as they were men under 50 years of age, with venereal histories, did not lose consciousness at the time of the "stroke" and had recovered in some meas-

ure, the attacks doubtless were due not to unilateral hemorrhage, but to syphilitic cerebral arterio-thrombosis which is often unequally bilateral. In none, was a typical image of paralysis of any particular group of laryngeal muscles presented, although the left cord in one was nearly, but not quite, motionless in the cadaveric position, the condition in two being an incomplete bilateral paresis which predominated in the larynx on the side of the paretic arm and leg, as evidenced by a feeble closure of the flaccid cords in a slanting position. Either an enfeeblement of the pharyngeal muscles, or impairment of articulation, or mild aphasia, was associated with the laryngeal paresis in one or another of this subgroup. One of them has remained under observation, receiving antisyphilitic treatment during a period of seven years and is now well.

The remaining three patients of this group were persons somewhat advanced in years who presented symptoms, in general, of cerebral degeneration, the paresis of the vocal cords being the cause only in part of the difficulty in speaking, although hoarseness and weakness of the voice were particular complaints. The laryngeal image was not typical and represented an unequal paresis of both cords rather than a complete paralysis of either cord. The local treatment was mildly palliative only, but proved far from useless, as the detergents and emollients applied by simple methods served to lessen the friction of vocalization and thus represented to the aged sufferers the difference between misery and contentment.

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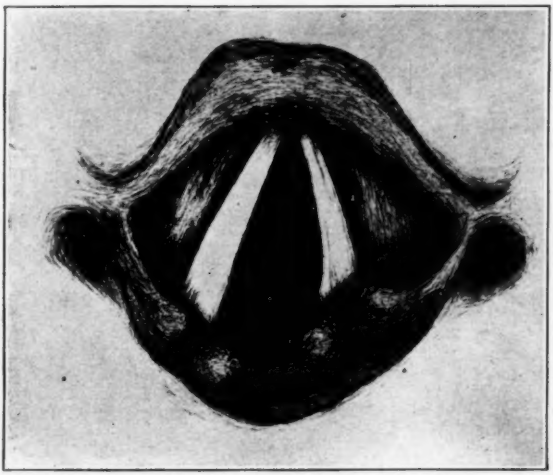

FIG. 1. The typical image of complete left recurrent paralysis. The vocal cord, in the cadaveric position, appears relaxed, narrowed, shortened and depressed; the arytenoid, "fallen in," not swollen.

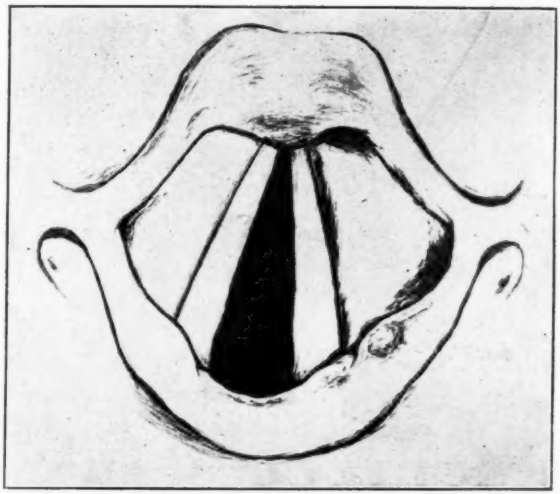

FIG. 2. The typical image of unilateral abductor paralysis. The vocal cord in the median or closed position. Arytenoid only slightly "fallen in," hence the cord is but little depressed or shortened.

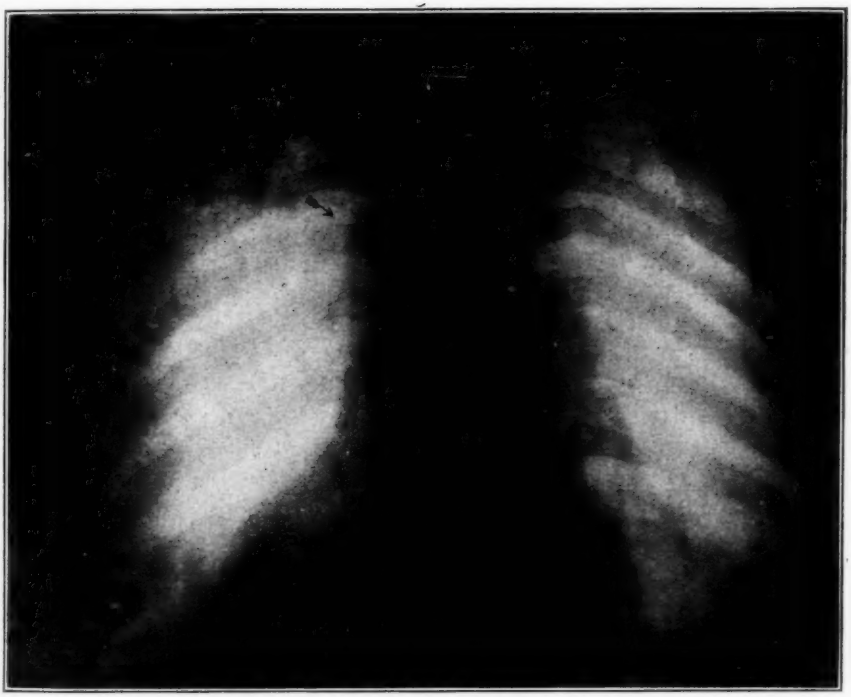

FIG. 3. Aneurysm, so small that the radiograph alone would have left it doubtful. Complete paralysis of the left vocal cord and exaggerated bronchial breathing present.

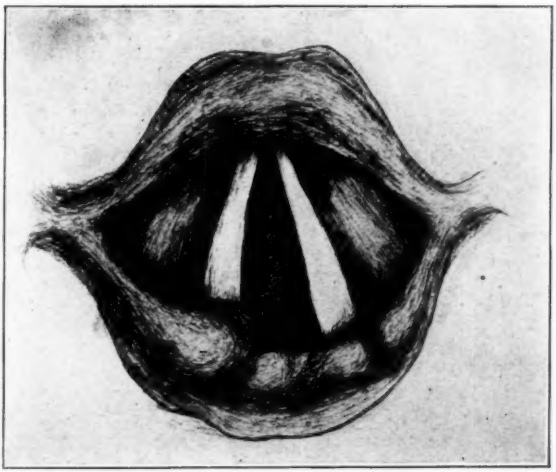

FIG. 4. A variation from the typical image of recurrent paralysis. The arytenoid is swollen.

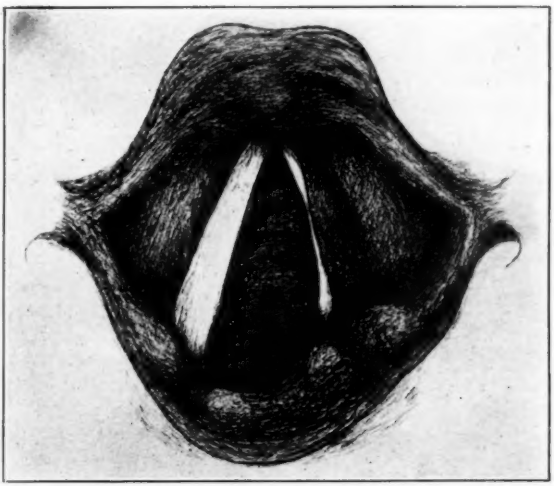

FIG. 5. A variation from the typical image of recurrent paralysis. The ventricular band is enlarged.

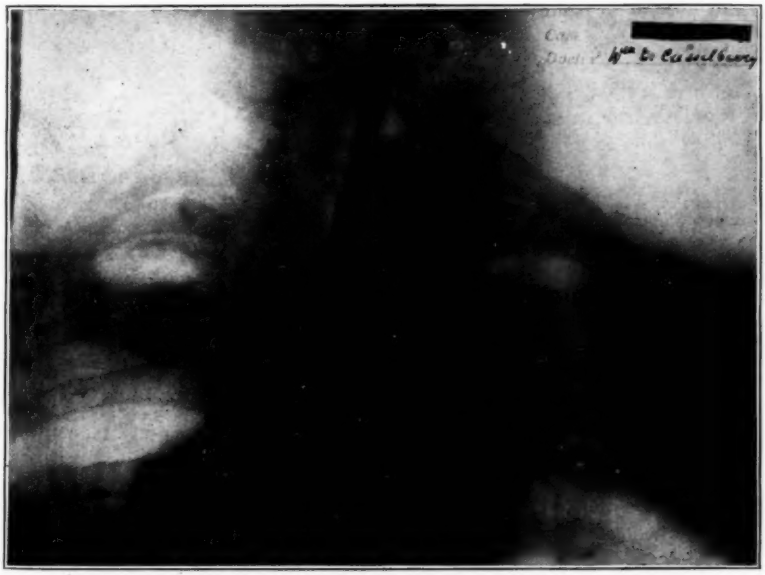

FIG. 6. Esophageal neoplasm. Differentiated from aneurysm by absence of pulsation when observed in the fluoroscope.

XXIII.

THE ETIOLOGY OF PARALYSIS OF THE RECURRENT LARYNGEAL NERVES OF PERIPHERAL ORIGIN.*

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Reviewing the history of this subject, we find that at a period centering about twenty years ago, much interest was shown in its study. Led by Prof. Hermann Krause in Europe and by the late Prof. Franklin H. Hooper in this country, excellent laboratory work was done and some important facts were demonstrated. Of the main questions at issue, however, many were unanswered, in spite of the earnest efforts of a number of well qualified investigators and of controversies which in some instances assumed the proportions of bitter feuds. Of late years not much has been advanced on the subject and that little has not been illuminating. Possibly the difficulty of the study and the lack of success attending its investigation have discouraged progress. Possibly again in view of the present popularity of other adjacent fields, the larynx has received less attention recently than it deserves.

The serious nature of recurrent paralysis and the not inconsiderable number of patients affected by it should certainly stimulate us to more earnest effort towards the better understanding and explanation of the causes which bring it about.

In many varieties of the affection the cause is clear. In others it is obscure, as witness the number of cases in which the diagnosis has either not been made or has been clouded by conflicting opinions as to the actual effect of the supposed cause.

I shall be able to do little more than call attention to some of the questions which the subject suggests and ask you to interest yourselves in taking up their investigation.

Indeed, in spite of all that has been written on the subject, it may be said that the study of this department is in its in-

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fancy, for it has not even been determined whether certain toxic paralyses of the recurrents are due to central causes, to changes in the nerve elements or to neuritis from severe surface irritation.

Recurrent paralysis may be either unilateral or bilateral, and only the posterior fibers of the nerve may be involved, as in abductor paralysis, or, the paralysis may be complete.

Shurly[1] quotes Avellis to the effect that of 150 cases, the cause was discovered in only 85. Of the above 150 cases, plus 19 collected by Sendziak,[2] 113 were males and 50 were females. In the same series of cases, the paralysis was found 46 times on the right side, 92 times on the left side, and 12 times on both sides. In 41 cases of Sendziak, 5 were right, 15 left and 21 bilateral. Unfortunately little reliability can be based upon these statistics. Three points become evident from them, however, as has been suggested by Lermoyez, namely:

The preponderance of peripheral paralysis over that caused by central lesions; the excess of cases in which the paralysis has been on the left side, and, finally, the greater liability of recurrent paralysis among men.

As to age, of 19 cases, 15 occurred between the 20th and 60th years. The youngest was a child of 2 years, the eldest was 59.

Sendziak claims to have collected 1017 cases, which he has classified according to the form of the paralysis, as follows:

1.	Abductor Paralysis.....	526	
2.	Paralysis of Recurrent.....	491	1017
1.	a. Unilateral Abductor Paralysis.....	283	
	b. Bilateral Abductor Paralysis.....	243	526
2.	a. Unilateral Recurrent Paralysis.....	397	
	b. Bilateral Recurrent Paralysis.....	94	491
		1017	1017

Among the earliest observations noted with regard to them was the fact that if the recurrent were injured, dyspnea was a prompt result. This was made apparent through the effect upon them of wounds of the throat and neck. Later on the possibility of paralysis from pressure was recognized.

Finally, within recent years, a few observers have called

attention to the occurrence of paralysis from toxic causes.

Many cases have been reported in which the movements of the larynx have been affected by peculiar conditions of the blood due to disease or to the ingestion into the system of certain well-known poisons. Thus gradually it has become apparent that paralysis of the recurrents of peripheral origin may be due to many causes.

The consideration of paralysis due to traumatism and to pressure are very interesting topics. Moreover the subjects are far from having been exhausted. Many things remain to be explained regarding both of them.

Paralysis due to toxic agencies, however, is a subject which has been hitherto little studied. It is replete with problems of many kinds and these again depend upon questions of surpassing interest. Few fresher or more attractive fields could be offered to the investigator, whether physiologist, pathologist or chemist.

In studying the reports of cases by various authors, the writer has been impressed with the belief that some of them were probably not cases of actual paralysis, but of ankylosis.[3] This is especially likely to be true in such diseases as typhoid, erysipelas and other conditions attended with severe local inflammation adjacent to the joint, or, as in rheumatism and gout, with inflammation of the joint itself.

The possibility of error in deductions based upon the statistics of reported cases of paralysis, some of which may have been wrongly diagnosed, must therefore be taken into account.

The possible myopathic origin of loss of motion in the larynx must also be remembered. This condition may be present in wasting diseases, muscular atrophy, rheumatism and carcinoma.

While first asking attention to the better known departments of the subject, it is to the toxic conditions that I shall finally bespeak especial attention and interest, believing that the time has come when light should be thrown upon the obscurities which surround them and hoping that ere long more complete and definite knowledge may be secured.

The causes of recurrent laryngeal paralysis of peripheral origin as far as known may be divided into three general groups, namely:

1. Cases due to traumatism, or laceration of the nerve trunks.

2. Cases due to mechanical pressure upon the nerve.

3. Cases due to toxic influences, either of the nature of poisons ingested into the body, or of toxemias or infections generated by disease.

As has already been said, careful distinction must be made between actual paralysis and simple loss of motion due to myopathic causes. Also, from loss of motion from ankylosis of the crico-arytenoid articulations.

Of the various causes of paralysis of the recurrents, we will first consider traumatism.

Laceration of the nerve trunk may occur in any part of its course and from a variety of accidents and the injury may be inflicted from without the larynx and trachea or from within them.

The list includes incised, gun-shot and punctured wounds of the neck, self-inflicted or accidental; injuries to the nerve made in the course of surgical operations done upon neighboring parts; foreign bodies in the larynx, trachea or esophagus and unsuccessful efforts made at their extraction; injury to the nerve from attempts at strangling and from blows upon the larynx.

Division of the recurrent happens occasionally in cases of cut throat. The possibility of its existence should be recognized before attempting the complete closure of such wounds, as the effects of sudden urgent dyspnea might of course be very serious.

Lefferts[4] has reported a case of paralysis of the right recurrent nerve due to a punctured wound made by the point of the blade of a pair of scissors.

The recurrents are liable to injury in operations for the removal of various tumors, especially of the thyroid gland. This is particularly true of goitre, as a number of recorded instances will attest.

In view of the increasing popularity of the operative treatment of goitre this should be carefully remembered.

Operations for the removal of new growths of the neck in which the necessary dissections approach the vicinity of the recurrents may easily be attended with injury to them, as also may attempts at the extirpation of diseased lymph nodes lying near to the larynx or trachea.

The writer has seen cases of paralysis of the right side of the larynx following extensive extirpation of the cervical glands in recurrent carcinoma of the right breast.

The position usually occupied by the left recurrent behind the inferior thyroid artery renders it liable to injury during ligation of the latter.

We will now consider the subject of recurrent paralysis due to mechanical pressure.

The list of known causes includes: Disease of glands and of lymph nodes; new growths; aneurysms; diseases of the heart; diseases of the pleura and scoliosis.

Comparatively few cases of recurrent paralysis have been recorded in any country. At present reliable statistics as to the relative frequency of these causes are not to be obtained. Accuracy of statement therefore is impossible.

Clinical experience seems to prove that this division presents by far the largest number of the cases that come under observation. Scientifically speaking, they are more interesting than the cases due to traumatism, for while some are relatively common, others are rare; and while some—fortunately, as a rule, the more common ones—are generally rather easy of diagnosis, there are no small number which are very difficult, so obscure in fact that a positive diagnosis with the means of examination at present available to us seems to be impossible.

Many cases hitherto obscure have of late years been made clear by the aid of the Roentgen ray. Perhaps nowhere among the varied uses of that splendid contribution to science have more brilliant successes been obtained.

We will begin with the diseases of the glands and of the lymph nodes.

That a diseased condition of the thymus gland has ever been the cause of laryngeal paralysis I have not been able to learn.

Disease of the thyroid gland, however, is a common cause, occasionally from malignant involvement, but far more often from goitre.

Remembering the anatomic relations of the thyroid with the trachea it is easy to understand how the recurrents might be encroached upon by almost any swelling of that gland.

The size of the thyroid tumor seems to have little effect upon the degree to which it may endanger the recurrents. Thus a very large goitre may cause little or no dyspnea. On the other hand, one of small size may prove fatal.

The most striking instance of this which the writer has even seen came to his notice when he was Assistant Pathologist to the New York Hospital some years ago.

A middle-aged lady of rather full habit, while shopping, was suddenly seized with urgent dyspnea. She was hurried to the New York Hospital, which was nearby, and within a few minutes after the onset of the attack tracheotomy was performed. Before the canula could be inserted, she died.

At autopsy made by Dr. Geo. L. Peabody, the thyroid appeared to be slightly enlarged. Upon exposing it by dissection, a goitrous tumor, parenchymatous in nature, was found, the base of which did not measure quite four inches in diameter. It was so situated with regard to the trachea, however, that both recurrents had been pressed upon sufficiently to cause paralysis or, as is more probable, spasms of the larynx and asphyxia.

Disease of the tracheo-bronchial lymph nodes has been the cause of recurrent paralysis in a number of recorded cases. These may have become enlarged from syphilis, tuberculosis, malignant infiltration, or other infections.

In a case seen by the writer at St. Luke's Hospital, New York, a child, 2½ years old, suffered from urgent dyspnea, requiring a prompt tracheotomy, which failed to give complete relief. Death followed in a few days. The child was found to have general tuberculosis, and the cause of the dyspnea was a large lymph node pressing upon the middle section of the trachea. Great obscurity must often attend the recognition of these cases. The fact of their possibility, the presence of a condition capable of causing them, the absence of other discoverable cause and above all the use of the x-ray may all aid in arriving at the truth.

Malignant disease of the thyroid, the esophagus, the neck and the mediastinum is a common, if not the most frequent, cause of recurrent paralysis.

In all of these but the esophagus it is generally due to direct pressure upon the nerve trunk. While sometimes due to direct pressure from disease of the esophagus, it may also in the latter case be caused indirectly through the extension of the disease when located in the upper part of the esophagus to the larynx or trachea, so that both recurrents are surrounded by cancerous masses and thus totally paralyzed. Or the

paralysis of the nerves may be direct from the pressure of degenerated cancerous glands upon them.

Sandby[5] has reported a case which has become famous for two reasons—first, because as a result of esophageal carcinoma adductor paralysis was first observed. Later this disappeared and a condition of purely abductor paralysis took its place. Finally the cords assumed the cadaveric position, thus proving the complete paralysis of the nerves. A second point of great interest in this oft quoted case is that it seems to contradict the so-called "law" of Semon as to the supposed vulnerability of the abductor filaments of the recurrent nerve.

In the minds of most practitioners, the discovery of fixation of the left side of the larynx is almost proof positive of the presence of an aortic aneurysm. Very often this view is correct. In the experience of the writer with the cases that have come under his observation, aneurysm of the aorta has been the most frequent cause of left recurrent paralysis. The mechanism of this is too familiar to require special explanation. It must be remembered that bilateral paralysis with its attendant dangers may be developed by the growth of the aneurysmal tumor in its late stages.

The position of the right recurrent with relation to the right subclavian artery is such as to render it vulnerable in case of aneurysm of that vessel. Such cases are very rare in comparison with those of left recurrent paralysis due to aortic tumor.

Aneurysm of the innominate has been said to be an occasional cause.

Cases have been reported in which recurrent paralysis has been caused by various diseased conditions of the heart, especially valvular disease in the form of mitral insufficiency and of pericarditis. By the use of the term "valvular disease" in this connection we are probably meant to understand the effects of it in the form of dilatation or hypertrophy, the increase in the size of the organ causing it to press upon the recurrents, as would be the case in pericarditis. Apropos of this subject, the case reported by Baumler[6] presents features of extraordinary interest.

A young man suffering from syphilis and an old mitral insufficiency lost his voice synchronously with the appearance of a pericardial exudation. Examination of the larynx showed a bilateral abductor paralysis. With beginning resorption of

the exudate the voice gradually returned, at first rough and hoarse. Two weeks later it became entirely normal and remained so until his death.

From its position with regard to the apex of the right lung the right recurrent may be pressed upon under certain morbid conditions of that part.

Thus, in fibrous phthisis, such cases have been found. They have also been caused by thickening of the pleura covering the right lung apex. It is said that the same thing has been seen in the case of the left lung.

Finally, a few cases are reported in which paralysis was attributed to penumonia, pneumothorax and pleurisy. That the paralysis was actually due to pressure and not the result of severe peripheral inflammatory conditions, remains to be proved.

By far the most interesting group of cases which come within the scope of this article are those due to toxic influences. They are less frequently observed than those caused by traumatism or by pressure, so that opportunity to study them has not so often been given.

Moreover, the physiologic and pathologic processes by which they are brought about are far too little discussed and much less well understood. It is proper therefore that particular attention should be called to them both by reason of their pathologic importance and because they offer one of the most interesting and valuable fields for original research to be found within the range of medicine.

The ingestion of certain drugs and chemicals is found to produce in some cases paralysis of the recurrent laryngeal nerves.

In like manner recurrent paralysis occasionally accompanies certain acute infectious diseases. If it be true that the poison acts directly upon the nerve-elements through some especial affinity of the one for the other, then the variety of toxics capable of so acting is remarkable, and it suggests a vulnerability of the recurrents to toxic effects which is, to say the least, unusual and peculiar. Other sets of nerves may be similarly acted upon, as in poisoning from lead, alcohol or diphtheria. The number of different toxins which have been said to affect the recurrents, however, seems unusually extensive.

The list of causes of toxic paralysis of the recurrents

should be divided into two groups: First, mineral and vegetable poisons, and secondly, toxins generated by certain acute infectious diseases. To the former belong lead, arsenic, antimony, copper, iodid of potassium, iodoform, and, it has been alleged, cyanid of potassium and phosphorus.

Recurrent paralysis due to other mineral poisons must be very rare. A few cases have been observed which seem fairly to have been due to the influence of arsenic, iodid of potassium and iodoform.

Those referred to antimony, copper, cyanid of potassium and phosphorus, are apparently of doubtful nature.

The first case on record in which the influence of lead upon the recurrents was observed by the aid of the laryngoscope was seen by Sir Morell Mackenzie[7] in 1865. He reported a case of complete paralysis of the right side of the larynx in a young man suffering from general lead poisoning. The paralysis disappeared after two months' application of electricity.

Our fellow member Sajous[8] in 1882 reported a case of bilateral paralysis of the larynx due to lead poisoning, and Seifert follows in 1884 with three well observed cases. A few others have appeared in literature up to the present time.

The cases reported in which other minerals have been thought to produce recurrent paralysis have been so few in number as to make deductions impossible. They suggest the possibility of coincidence quite as strongly as they do that of cause and effect.

Paul Heymann[9] collected over 50 cases of mineral poisoning of the larynx. He found lead to be the most common cause. Usually the abductors were affected.

Among the vegetable poisons, the following have been alluded to in the literature of the subject: alcohol, opium, morphin, belladonna, atropin, cannabis indica, cocain.

Among the vegetable poisons mentioned, alcohol properly heads the list. Hippocrates[10] calls attention to that form of aphonia of the drunkard which comes upon him in his cups and does not disappear when he has become sober. Several good authorities have quoted the coexistence of recurrent paralysis with alcoholism. Cases have also been reported as being due to opium and to belladonna, and it has at least been suggested that similar results may arise from cannabis indica and from cocain.

The questions raised by the action of the drugs and minerals just described upon that always peculiar pair of nerves, the recurrent laryngeals, are perplexing. In what manner do they inhibit the action of these most active parts?

1. Is there any truth in the supposition that there is an "elective affinity" existing in these particular nerves in favor of certain drugs through the influence of which the one exercises an inhibitory effect upon the other?

2. Does the drug produce changes in the nerve tissue itself which result in the loss of its activity?

3. Is the lesion a neuritis, induced by pressure from congestion of neighboring parts or otherwise?

5. Are the effects upon the recurrent due to central causes, the latter induced by the drug?

Turning now from the mineral and vegetable poisons which may affect the recurrents, we take up the consideration of the alleged results produced by the toxins of some of the acute infections:

These include typhoid fever, acute articular rheumatism, influenza, diphtheria; typhus fever, acute croupous pneumonia, puerperal fever, erysipelas, measles, scarlet fever, gonorrhea, serum therapy.

Of 73 cases collected by Sendziak, 26 occurred in typhoid fever, 18 in acute articular rheumatism, 11 in influenza, 9 in diphtheria, 3 in typhus fever, 2 in croupous pneumonia, and 1 each in puerperal fever, erysipelas, measles, scarlet fever, gonorrhea and the use of serum therapy.

Of the infectious diseases in which recurrent paralysis may be found typhoid fever seems decidedly the most common. The paralysis may be unilateral or bilateral. It may occur early in the course of the disease, but it generally appears towards its close, during the 3d or 4th week.

Osler,[11] agreeing with the generally received opinion, attributes it to a neuritis of the recurrent and states that the general activity of the toxins in a given case has more influence upon the nervous system in causing the neuritis than has the temperature. In other words, the neuritis is not so apt to be developed in a case where the temperature is running a high course as it is in one in which the typhoid intoxication is profound.

Laryngeal paralysis in a severe case of typhoid is, of course, an exceedingly grave complication, although in the milder

cases of fever and where the paralysis is unilateral it is not so dangerous.

Regarding the cause of the paralysis, the question again arises as to whether, after all, the nerve irritation is not brought about by the violent surface inflammation, commonly met with in and around the larynx in this disease.

While most writers state that the paralysis of the recurrents in typhoid is generally due to neuritis, certain ones believe that the loss of motion is sometimes myopathic. Degeneration of the laryngeal muscles may take place in like manner with the degeneration of the abdominal muscles in typhoid.

Morris Schmidt[12] says that recurrent paralysis has occurred in typhoid patients suffering from complicating pleuro-pneumonia, glandular swelling and muscular degeneration.

May it not be possible that some of the instances reported as being due to typhoid toxemia have, after all, been caused by such complicating conditions?

In typhus fever, as in typhoid, recurrent paralysis may supervene. It is said to be more frequent in typhus than in typhoid; it is likely to occur at an earlier period of the disease and to be of a more serious nature.

In acute articular rheumatism recurrent paralysis has been found in all its phases: unilateral, bilateral, abductor and complete.

Here again the causes are variously stated, neuritis and rheumatic affection of the laryngeal muscles having the preference.

Clinton Wagner and others have reported cases of gouty inflammation of the crico-arytenoid articulation with perichondritis, necrosis, abscess and sloughing of the arytenoid cartilage. The possibility of such a case should suggest the possibility of rheumatic affection of the laryngeal muscles and admit of the assumption that not every case of loss of motion in the larynx is necessarily neuropathic.

Morris Schmidt[12] says that he has not infrequently seen recurrent paralysis following influenza. Other writers have reported a few such cases. This does not seem improbable in a disease capable of affecting so profoundly the nervous system in general.

Morris Schmidt[12] also calls attention to the fact that in diphtheria it is still questionable whether the resulting paraly-

ses are due to central or to peripheral causes. In the opinion of the writer it is not necessary that fixation of the larynx following diphtheria should be due to paralysis at all, since under conditions of inflammation as violent as those often found in laryngeal diphtheria, ankylosis of the cricoarytenoid might easily be produced. In several cases seen by the writer of unilateral loss of motion present many years after an attack of diphtheria this has seemed to be the true explanation. The question is rendered still more difficult, however, when we remember that following a genuine paralysis the joint may become ankylosed through disuse.

Apparent paralysis of the larynx may also accompany the severe infections of the throat non-diphtheritic in type, as for example, the staphylococcus and the streptococcus. It seems more probable that the loss of motion is due rather to the effects of inflammation upon the muscles and joints of the larynx than to its effects upon the recurrents.

One case each is reported of recurrent paralysis from erysipelas, puerperal fever, scarlet fever, measles, serum therapy and gonorrhea. In all but the first the paralysis was unilateral.•

The last named case has been elaborately described in an extensive and scholarly article by Lazarus.[13]

With regard to toxic paralysis, some very interesting questions arise.

1. If it be true that there is such a thing as a "selective affinity" between the recurrents and a given poison, in a case of poisoning we would expect to find both recurrents affected. In point of fact mineral poisons seem almost invariably to affect but one.

Again, if the toxins of infection cause paralysis by exciting a recurrent neuritis, we would expect the result to be bilateral, whereas a considerable proportion of such cases have been unilateral. Both of these facts seem to contradict the affinity doctrine.

2. Is it possible that toxins, whether inorganic or organic, produce special structural changes in the nerve tissue itself, which result in recurrent paralysis.

For the answer to this question we must look to the pathologist, although it would seem that simple inflammation of the nerve would be all that was necessary to inhibit its activity.

3. Are the effects of toxins upon the recurrent due to central causes? Here again the fact that bilateral paralysis is rare and unilateral the rule would throw doubt upon the probability of an affirmative answer.

4. Is the lesion a neuritis, induced by pressure from congestion of neighboring parts or otherwise? It seems probable that in many cases it is, since this would explain the presence of recurrent paralysis from so many different varieties of causes, most of which are associated with congestive conditions of the throat, and for its occurrence in so many different forms.

It is easy to suppose that an inflammatory condition severe enough to cause a crico-arytenoid ankylosis might cause a paralysis of nerves lying as near the surface as do the recurrents.

It is impossible to escape the conviction that some of the cases of recurrent paralysis found accompanying violent inflammations of the throat have been due to the effects of superficial irritation.

Nothing is more common than to find a paralysis of the posterior arytenoideus muscle accompanying an acute laryngitis. The position of this muscle near the surface and at a part of the larynx where the inflammatory conditions are apt to be most intense, would render its nerve filaments peculiarly liable to irritation and injury.

Percy Kidd[14] reported a case of bilateral paralysis of the larynx, cadaveric position, following a severe laryngitis, lasting about three weeks and ending in recovery.

Such cases are not unique. If they suggest anything, it is that surface irritation may be the cause of recurrent paralysis. In many of the diseases in which recurrent paralysis is reported to have occurred, inflammation of the throat is a marked symptom.

If it can be caused by the irritation of a simple laryngitis, it is certainly possible that the still more violent inflammation accompanying severe acute infections may produce it. Doubtless among both groups of toxic causes there are some which act in this way.

These are some of the questions that remain to be explained and finally answered. If my presentation of them has been imperfect and my deductions inadequate, it must be remembered that they have thus far baffled the efforts of all

who have attempted their solution. With the advance of knowledge of the nervous system in general and with the increased accumulation of clinical material it is to be hoped that better progress may ere long be made.

A study of the literature of this subject shows that it leaves much to be desired. Considering the large variety of conditions which may cause it and the vast number of the cases which many of these conditions create, as for instance, traumatism, tumors, cardiac diseases, tuberculosis, typhoid, diphtheria, influenza, etc., it is strange that relatively so few recurrent paralyses have been recorded. Doubtless more careful observation, aided by laryngoscopic examination, would demonstrate many additional ones. Here, as is the case in many other departments, many of the histories have been too incompletely recorded to be of any value.

The latest writer, Sendziak,[2] claims to have found 1017 cases, 201 of which he says are his own. Where he found them he does not state. Unfortunately he does not give the references to them, so that his statistics cannot be verified, nor can the articles from which he derived his material be studied. His own articles being published in the Polish language and in obscure provincial journals, makes them almost impossible of access.

The article of Lermoyez is classic and well worthy of study. Paul Heymann's[9] monograph on recurrent paralysis, due to the effect of poisons, is excellent. It contains a full and very valuable bibliography.

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XXIV.

THE ETIOLOGY OF OTOSCLEROSIS.*

By Thomas J. Harris, M. D.,

New York.

The secretary is to be congratulated on putting a subject as important as this on the program for discussion this year. It is my only regret that the duty of presenting the first paper in it should devolve on one so incompetent. Twenty years ago we heard and read little upon this potent cause of deafness. The textbooks of that day mentioned a form of middle ear disease known as dry or hyperplastic catarrh, but the impression was given that it was met with only infrequently and was of little significance compared with the common hypertrophic variety. Since that time otologists, especially those abroad, have busied themselves with the subject and an inspection of the literature of the past two decades will show many valuable contributions thereto. There is today in spite of all this, much unmerited ignorance and too often it is grouped clinically with the other non-suppurative affections of the middle ear.

Any intelligent consideration of the causes of otosclerosis demands a clear understanding of the exact pathologic process which is producing the affection. This since Politzer's valuable paper, later reinforced by Siebenmann's work, has commonly been held to be a rarefying process of the labyrinthine capsule or the region in the vicinity of the oval window either internal or external to it or both. This is regarded by Politzer to be a primary affection. That such a condition at times exists cannot be gainsayed as has been abundantly shown on the postmortem table. That it is the usual condition in the cases which we are accustomed to regard clinically as otosclerosis, is, however, decidedly open to question. The grounds for this radical departure from what has been a generally accepted position of all writers,

*Read at the meeting of the American Otological Society at Atlantic City, June 24, 1908.

are clearly set forth in Lucae's recent treatise on the subject entitled "Die Chronische Progressive Schwerhörigkeit."

Lucae calls attention to the fact that von Troeltsch was the first to make use of the term sclerosis, and while the latter does not specifically mention it, he in all probability based his employment of the term on the careful description of a group of cases by Toynbee which he characterized as cases of rigidity of the lining membrane of the ear. Adopting Toynbee's idea of the pathology in such cases and defending his position at much length and by the citation of numerous cases of his own, Lucae advances the theory that a previous inflammatory condition, either the ordinary hypertrophic form or that of suppuration, is at the bottom of the great majority of all cases of sclerosis. Such a condition, especially one of transient suppuration occurring in childhood or infancy, as it often does, can most easily be lost sight of at the period when the symptoms of the disease are first noted. Toynbee's description of these so-called cases of "rigidity" of the mucous membrane of the ear is deserving of being quoted in full: "The mucous membrane of the tympanum may be subject to chronic inflammation, complicated or not with rheumatism, at any period of life, and, if neglected, this is liable to terminate in a rigid state of the membrane; so that the ossicles become bound together more firmly than is natural. In advanced years, this rigid condition seems to take place without any symtom of inflammation. The membrana tympani also partakes of this rigid condition, and, what is of far greater importance, the base of the stapes may become much more firmly fixed to the border of the fenestra ovalis than is natural. As a consequence, the membrane of the fenestra rotunda and the fluid of the labyrinth participate in this fixed condition. This affection is less prevalent in the young than in the adult, in whom it is liable to occur after repeated attacks of cold, whether with pain or not. It is, however, most frequent in persons advancing in life, and may, in fact, be considered as the disease which causes deafness in advancing years. The generally received opinion that in this kind of deafness the nervous system is at fault, is manifestly incorrect, as proved by the symptoms and by the mode of relief found beneficial." In addition to this condition so clearly set forth, Toynbee describes two others

giving rise to similar symptoms. First, membranous bands in the tympanic cavity. "These may arise in any part of the cavity connecting together the ossicles and attaching them to the walls of the tympanum and to the drum membrane." The stapes is wont to be bound down to the promontory and thus a species of ankylosis produced. Toynbee is of the opinion that these bands are the result of the effusion of fibrin from the membrane or from the adhesion together of the already hypertrophied membrane.

The second condition is that of ankylosis of the stapes, so well known to all aurists since his day. In his opinion, however, this ankylosis permits of various gradations according to the stage of the disease. At first it is only partial without expansion of the base of the stapes, depending on a rigidity of the capsular ligaments. This is the earliest form. A second stage is the expansion of the articular surfaces without changes in the bone. This was found in 49 of the 136 specimens of ankylosis described in his catalogue. The later stages represent hypertrophy of the entire stapes base. Here it is often impossible to remove the crura from the oval window without breaking, and finally we may meet in the most advanced cases, actual deposit of bone.

These classical views of one of the earliest workers in our specialty, we have taken the liberty of quoting at some length because of their direct bearing on the question of etiology under consideration and because they serve without doubt as a basis for the views which have been set forth by Lucae in his recent book just referred to. Lucae is prepared to admit that a certain percentage of ear cases will fall in the group of primary disease of the labyrinthine capsule but believes that careful inquiry as to a previous ear condition and the employment of the more accurate methods of diagnosis, will serve greatly to reduce the number. The opinion of so competent and experienced an observer, is worthy of our most thoughtful consideration. While not prepared perhaps to subscribe to it at present in its entirety, we feel that it represents in the main a reasonable view of the subject, one that offers hope as far as prognosis is concerned where hope formerly could not be entertained and one entitled to be applied to all cases of this class which present themselves to us for treatment.

We would repeat then that in our opinion otosclerosis

represents not one but several pathologic conditions. Viewed from this standpoint the question of cause or causes becomes a broad one. Inasmuch as there seems in the majority of cases to be a precedent middle ear catarrh, it may be said that the same causes will tend to produce both. It is generally agreed that the most common cause for the former is disease of the oro- and epi-pharynx, and that the extension to the ear is wont to take place through the Eustachian tube. To this view we have not the least wish to take any exception. Indeed, experience only impresses upon us the close relation which exists between the two localities, but as to the equally popular teaching of the importance to be attached to affections of the nose in their effect on the ear, we have still to be convinced. At the same time we are willing to admit that marked obstructions in the nares can be accountable for a certain number of ear cases. Their number in our judgment is, however, small in comparison with the number due to other cases. Moreover it must be constantly borne in mind that we continually meet patients with complete nasal obstruction due to polyps or other neoplasms without any aural symptoms whatever. Indeed, we would go further and say that we believe that in many instances where is well defined throat lesion, the ear disease is not dependent upon this but that there is a common cause producing both.

In line with what has just been said regarding the relation of otitis media chronica hypertrophica to sclerosis and bearing on the question of a common cause for affections of the throat and ear, we may be permitted to refer to the statistics of 21 cases of undoubted sclerosis seen recently in private practice. Many of them had undergone operations upon the nose or throat without benefit before coming to us, and what is of particular interest, in not one of them at the time of our examination was there what might be described as a gross nasal lesion, while in most there was no complaint addressed to the nose at all. Yet in 15, or over 66 per cent, there was a clear history of repeated colds extending over several years. This complaint of repeated colds is the most significant feature of the history of many of these cases and one upon which the greatest emphasis is to be placed. The cause in our judgment in the majority of instances is not to be found alone or chiefly in the nose,

as is shown by the failure to relieve by nasal treatment in the cases just quoted and in many others with which you all are acquainted. This cause without question varies in the particular case. Rheumatism and gout have always been regarded as among the more common contributing causes. Our experience with gout in this country is of necessity limited and we do not recall finding many cases where a well-established rheumatic history was obtainable. Rather do we think that emphasis should be put upon that condition which is often in the lay mind confused with rheumatism and which very possibly is at the bottom of many cases, i. e., lithemia or collemia as described by Haig, that condition of the system where a state of autointoxication exists due to the retention of certain products of excretion in the blood. We know that this accounts in no small degree for many cases of throat difficulty and there is no question in our own mind that a similar cause operates not infrequently to set up or aggravate trouble in the ear. Another widely acting agent in exciting both pharyngeal and aural inflammation and one that we have yet to grasp the full significance of, are bacteria. Most writers who have looked into the subject, are beginning to believe that there is no difference etiologically between an acute catarrhal otitis and an acute suppurative otitis except in the virulence of the bacterial invasion. In addition to rheumatism, many other constitutional diseases have been suggested as exciting causes. Among these may be mentioned tuberculosis, syphilis, struma, arteriosclerosis, and trophoneurotic changes which occur in tabes and allied affections. All can and do undoubtedly exist in connection with otosclerosis but the direct causal relation is yet to be shown except as any profound constitutional disturbance must affect every organ of the body and render it more liable to disease. The female sex is especially prone to the disease, which would seem to suggest the probability that a highly organized nervous system, such as is wont to be found in women, is an important predisposing factor.

Age would appear to have some bearing upon the onset of the disease. At least it makes its appearance in the majority of cases in early manhood and womanhood. In 733 cases reported by Lucae, it developed in 74 per cent between the years of 21 and 50. In 87 per cent of these,

the affection was double sided pointing to the general character of the malady.

Pregnancy has been advanced as a possible cause. While it does undoubtedly occur that marked flagging of the hearing occasionally is noted at that time, it can only be regarded as an instance of the now well recognized intimate relation between the sexual and sensory systems.

More striking than all other causes save the one of throat disease, is heredity. There can be no question that heredity plays an important role in otosclerosis. In 641 cases where records on this point were kept by Lucae, 235 or 37 per cent gave such a history, while Bezold puts it as high as 52 per cent in his cases. When it is considered how difficult it is to obtain accurate information regarding this due to the early death or separation of the parents or grandparents, there can be little question that the figures of Lucae are too low rather than too high. Highly significant as bearing on the question of the frequency of a preceding chronic catarrhal otitis in the majority of cases, is the fact that of the 235 cases where heredity was shown, 77 per cent had demonstrable pathologic changes in the tympanic cavity. This would seem to prove conclusively that inherited otosclerosis does not differ at all in this particular from the non-inherited variety. Quite in line with this observation and serving at least as a most ingenious explanation for this widely recognized peculiarity, is the theory of von Troeltsch that the family likeness is to be found as far as the ear is concerned in a uniform narrowing of all its measurements predisposing as soon as any inflammatory process has appeared, to an early throwing out of adhesive bands and the binding down of the ossicles in consequence. Siebenmann has also advanced the view that there exists an abnormal post-embryonic developmental process. Others have tried to show that the disease is especially wont to develop in those who are anemic or disposed to worry. Certain it is that some general underlying predisposing cause is needed to explain so widespread and characteristic a feature of the disease.

A careful and painstaking review of most of the recent articles on the subject, leads us to feel that not enough attention has been paid to this phase of the question and in order that more definite and accurate data may be secured,

we would urge that in all our future cases we endeavor to secure the fullest information possible on every point which may however remotely assist in arriving at an authoritative answer to the all important question in the consideration of any disease, namely, what is the cause?

XXV.

PATHOLOGY AND PROGNOSIS OF CHRONIC PROGRESSIVE HARDNESS OF HEARING—OTOSCLEROSIS.

By W. Sohier Bryant, A. M., M. D.,

New York.

This title assigned to me is somewhat misleading; I should prefer to substitute "Pathology and Prognosis of Chronic Interstitial Otitis." I am glad to have the opportunity to express my views on this subject, since I think confusion has arisen by the loose way in which the terms otosclerosis and chronic progressive hardness of hearing have been used by various authors. I use otosclerosis to denote an interstitial inflammatory sclerotic condition of the ear especially characterized by vascular changes and usually located in the tympanic cavity or in its walls. The term otosclerosis is in no way expressive of the vascular ostitic lesions to which it is often applied. On the other hand, I use the term chronic progressive hardness of hearing to denote a class of ear affections due to changes in the middle ear or its walls, these changes being independent of ulcerative processes but characterized by chronicity and a tendency to increasing deafness. In exceptional cases the hearing appears to remain stationary for long periods. Most of the cases, all except the most advanced, may show arrest of the process and a degree of improvement under proper therapeutic and surgical management. Some of the synonyms used for chronic interstitial otitis are: (1) dry chronic middle ear catarrh (Schenke); (2) otosclerosis; (3) chronic progressive deafness; (4) otitis media insidiosa; (5) rarefying ostitis of the labyrinth capsule. None of these terms is good. "Dry middle ear catarrh" does not express the pathologic condition. "Otosclerosis" is an expressive term but it has been ruined by its erroneously restricted application. "Chronic progressive deafness" has also been misapplied. "Otitis media insidiosa" is a rare form of chronic interstitial otitis and the term is not sufficiently

comprehensive. "Rarefying ostitis of the labyrinth capsule" is inadequate since it defines only a local terminal condition of the general pathologic process. My only excuse for mentioning these different terms is an attempt to explain away a misunderstanding in order to go back to the fundamental etymopathologic meaning of the words. For the general pathologic condition we are considering I would prefer to use the term chronic interstitial otitis with a special adjective referring to the parts affected in any one particular case, as otitis interstitialis chronica membranae, otitis interstitialis chronica ossiculae, otitis interstitialis chronica promontorii et fenestrae ovalis, otitis interstitialis chronica labyrinthi.

Coming back now to our subject, the results of clinical observation and treatment show that chronic interstitial otitis is due to middle ear, tubal and nasopharyngeal disturbances in many cases and to constitutional causes in others, or to all combined. The nasopharyngeal disturbances seem to work in a reflex way through the complicated nerve connections of the tympanum. Ever since I took up our specialty I have paid a great deal of attention to this subject and the overwhelming evidence of my clinical observation and experience together with laboratory work have forced me to accept the following views:

I. PATHOLOGY.

The pathology of the condition under discussion is dependent upon vascular disturbances affecting the blood supply of the tympanic cavity and walls, with resultant trophic changes. These pathologic processes have been described by many authors. The alteration in the blood supply causes either a hyperemia or anemia. Either of these conditions may be primary, but the anemia alone can be secondary. The primary anemia is of vasomotor origin and is usually found in young females. It has been described under the name of "otitis media insidiosa," a rare condition. The disturbance of the blood supply may be general or confined to one or more of three regions: (1) The drum membrane; (2) the major ossicles; (3) the stapes and labyrinthine wall. The changes are in most cases unequally distributed throughout the tympanum and in some cases may be sharp-

ly defined and limited to very small areas. Different stages are usually present at the same time. For instance extreme atrophic thinning, degeneration, calcification and fibrous thickening may be present in the same membrane. These circumscribed areas of different processes give a mottled appearance to the tympanic membrane.

There is nothing peculiar to the trophic changes caused by the altered blood supply of the middle ear. These changes are identical with the results of similar causes in other parts of the body, namely, congestion, vascular proliferation, infiltration, interstitial inflammation, hyperplasia, contraction, compression of blood vessels, anemia, degeneration, calcification, vascular ostitis or osteoporosis and hyperostosis. The peculiar feature exhibited by the middle ear is the very limited and sharply circumscribed area of the changes. The reason why similarly circumscribed changes are not found in other regions is that nowhere else do similar anatomic relations occur. The almost terminal arterial supply of the tympanum and its walls, where the mucoperiosteum of the tympanum admits of a blood supply running in one plane only, tends to render the blood supply subject to extreme variation over small areas. This tendency is enhanced by the peculiarly complicated arrangement of the tympanic plexus of sensory, motor and sympathetic nerves. Similar anatomic arrangements are not found anywhere else in the body. The tympanum has another peculiarity; wholly independent of central influences, it can control its own blood supply through its ganglionic connections. This control is brought about by the direct connection of the carotid and the minor vascular plexuses of the tympanic system through the intervention of the sensory nerves of the drum cavity. The nerve ganglia connected with the ear make it more subject to vasomotor changes due to local causes, than any other regions can possibly be. Among the most important causes of hyperemia are infectious processes of all grades whose resultant cicatricial contractions produce local anemia.

Because the mucous membrane is the most liable to vascular change, the pathologic changes under consideration affect this first; next they affect the submucous or periosteal layer, and last the bone. The pathology is exceedingly simple, but owing to the variety of structures involved and the

location of the processes the resultant subjective and objective effects are quite varied. Certain areas of the tympanum, the oval window, promontory and drum membrane are most often affected because these areas are more subject to reflex vasomotor and other vascular changes. These parts also are more apt to attract attention because of marked symptoms.

The congestive stage of the process is accompanied by vascular increase and later cellular infiltration; increase of fibrous tissue results, the contraction of this new connective tissue shuts off the blood supply and nutrition suffers with signs of degeneration and loss of pliability of the connective tissue. After the congestive stage is passed the mucosa, mucoperiosteum and drum membrane have thickened and become grayish white. Later there may be a deposition of lime salts in the fibrous and periosteal layers. Osteoporosis follows the vascular ostitis or congestive stage in the bone. The diminished blood supply of the periosteum and bone caused by fibrous contraction and shutting of the vessels results finally in absorption of bone or osteoporosis and in the end hyperostosis occurs following the bone absorption and in conjunction with it.

Gross examination and histologic findings of the tympanum show one or more of the above mentioned pathologic conditions which take the following forms: (1) The congestion of the drum membrane due to an engorgement or an increase of the vessels of the mucosa, later similar changes of the membrana propria; (2) anemia of the drum membrane due to contraction or compression of the vessels; (3) opacity of the drum membrane due to a loss of transparency of the mucous or fibrous layers from cellular infiltration, fibrous thickening or degenerative processes and calcification of the connective tissue; (4) thickening of the drum membrane accompanied by opacity and due to the cellular infiltration and an increase of the connective tissue element; (5) thinning of the drum membrane due to atrophy of the mucous layer and of the membrana propria; (6) functional impairment of ossicular sound transmission from congestion and edema of the ossicular articulations, especially of the orbicular articulation of the stapes; (7) contraction of fibrous adhesions of ossicles, drum membrane or tympanic walls. These adhesions are due to hypertrophy

or contraction of the normal mucous membrane, reduplications or to cicatricial adhesions; (8) rigidity of the articulations of the major ossicles and stapes due to atrophy and degeneration of fibrous tissue; (9) congestion and cellular infiltration of the promontory similar to the corresponding changes in the drum membrane; (10) functional rigidity or impairment of motion of the stapes without ankylosis due to congestions and cellular infiltration; (11) rigidity of the stapedio-vestibular articulation and fibrous ankylosis due to contraction of new fibrous tissue; (12) bony ankylosis of the foot plate of the stapes due to hyperostosis of the stapes and oval window; (13) vascular ostitis and osteoporosis of the ossicles and tympanic walls; (14) hyperostosis of the ossicles and tympanic walls; (15) hyperostosis of the labyrinthine walls, encroaching upon the labyrinthine spaces and interfering with the functions of the membranous labyrinth.

The bone changes begin with a thickening of the mucoperiosteum due to increased vascular formation and cellular infiltration. The nutrient vessels of the bone undergo similar changes. Later the new connective tissue formed from the infiltrating cells contracts, compresses the vessels and lessens the blood supply and interferes with the nutrition of the bone causing the formation of lacunae. Slightly later in the process but concomitant with the vascular ostitis the periosteum commences to form new bone in the form of irregular hyperostoses. The bone changes are most apt to commence in the neighborhood of the oval window and are usually found in the outer labyrinthine wall.

The pathology is founded on the etiologic basis of primary and secondary disturbed circulation, due to local or general causes. Because of the notoriety certain authors have brought upon these conditions and on account of the nomenclature erroneously applied to them, the pathology of the bone changes taking place about the stapes and in the labyrinthine capsule and other parts of the tympanic walls and temporal bone deserves special mention. Many authors have been surprisingly unanimous in their observation concerning the pathology of the condition rightly called rarefying ostitis. When, however, it comes to the steps which lead to this final condition, there is a great variance of opinion which increases when the primary pathologico-etiologic factor is under consideration

Many authors maintain with plausible arguments that this condition is a true bone syphilis; others equally plausibly disprove the presence in many cases of syphilitic infection. What then is the explanation? If we compare the progressive pathologic steps and the histologic findings with the course and result of syphilitic bone inflammation, we find an absolute identity of changes and histologic pictures. First there is an engorgement of the blood vessels followed by interstitial infiltration of round cells causing an increase of connective tissue elements. Then contraction of the connective tissue takes place shutting off the blood supply, next vascularization and vacuolization of the bone commences and finally new bone is formed.*

We know that the tympanum is subject to vasomotor changes which finally cause diminution of blood supply and atrophic changes. We know from frequent observation that the oval window and promontory are especially liable to chronic congestive states. Why should not the congestion produce the same result on the promontory as elsewhere, and why should not this result take the same form and produce the same alterations as similar vascular changes do in other bones? In default of a more logical primary pathologic cause for vascular ostitis of the labyrinth capsule, I believe it may perhaps be due to syphilis in some cases, but in the great majority of cases I believe it is due to altered blood supply of the lining mucoperiosteum of the inner wall of the tympanum independent of syphilis. Chronic interstitial otitis, including cases previously described under otosclerosis and spongifying ostitis of the labyrinth capsule, may follow, as we have seen, suppuration and infection as well as vasomotor changes and so-called "chronic middle ear catarrh," and therefore chronic interstitial otitis is a comparatively common affection. The pathologic changes in chronic interstitial otitis following suppuration are due to the diminished blood supply caused

*Schenke. Anatomischer Beitrag zur Frage der bei dem trockenen chronischen Mittelohrkatarrh (Sklerose?) vorkommenden Knochenerkrankung des Schläfenbeins (chronische vaskuläre Ostitis Volkmann), etc. Arch. f. Ohrenheilk., Vol. 53, p. 68, and plates, 1901.

Haberman. Zur Pathologie der sogenannten Otosklerose. Archiv f. Ohrenheilk., Vol. 60, p. 37, and plates, 1903.

Parrot. La Syphilis Héréditaire. Masson, Paris, 1886, plate 20, fig. 1.

by interstitial infiltration brought on by the suppurative inflammation which ends in fibrous contraction. That rarefying ostitis should be hereditary is natural, since it depends on nervous instability and morphologic irregularities, qualities which are hereditary to a degree.

II. PROGNOSIS.

The prognosis of chronic interstitial otitis is foreshadowed in the pathology. Without treatment the prognosis indicates a more or less gradual progressive deterioration of hearing. Under treatment the prognosis is favorable for improvement or prevention of further loss of hearing unless the pathologic changes are already far advanced. This improvement is possible because the fundamental causes of the altered blood supply which is the primary cause of the interstitial otitis are either local, nasopharyngeal or general conditions amenable to special treatment. If the blood supply is rectified before tissue changes have taken place, no sign will be left of the disturbances. The prognosis for the revitalization of degenerated tissues is dependent upon the prognosis for the improvement in the blood supply and our power to regulate the local vasomotor functions. If vasomotor regulation is established and the blood supply is corrected after tissue changes have taken place, the prognosis depends upon the ability of the patient to respond to the improved condition and to overcome the existing pathologic changes. The improvement in the ear following the improvement of the blood supply may extend to a complete restoration of function and return of the normal appearances of the drum membrane and ossicular chain. The prognosis is good if the congestive stage has not been passed, and good also after the progressive tissue changes have commenced up to the time the degenerative changes are very far advanced. The prognosis for the improvement and correction of adhesions by cutting and stretching is good. The prognosis for overcoming ankylosis is good if bony ankylosis and extreme degenerative states have not taken place. In bony ankylosis the prognosis is absolutely bad.

These previously published cases are illustrative of the prognosis in the more doubtful or advanced conditions:

1. Case No. 14229. A man, aged 67. Deafness commenced eight years ago; hearing has been very bad for three years; this condition was possibly aggravated by business failure. Hearing variable. Drum membrane whitish and opaque. Position, contour and light reflex normal; malleus movable. Politzer's acoumeter heard in the right ear at 4 inches, in left ear at 3 inches. Loud conversation heard in right ear at 6 inches, in left ear at 32 inches. Watch not heard in either ear. Bone conduction much shortened. Low limit for tone perception by bone, 128 single vibrations. Low limit for tone perception by air conduction above 256 single vibrations which were not heard by air. High notes well heard. Treatment by general hygiene and regulation of blood supply of the middle ear. Six months later, Politzer's acoumeter heard in right ear at 7 feet; watch at 20 inches; in left ear, Politzer's acoumeter heard at 4½ feet. Improvement has continued now for a period of four years.

2. Case No. 13083. A man, aged 47, had noted deficiency in hearing for eighteen years. Drum heads good color, fair contour and position, light reflexes very small. Nares partially occluded by hypertrophies and irregularities. Fossae of Rosenmüller partially closed by adhesions. Tubal mouths slightly obstructed by thickened mucosa. Watch heard in right ear at 15 inches; in left ear at 4 inches. Astringents and irritants applied to nasopharynx. Three years later watch heard at 48 inches in left ear and 84 inches in right ear. Improvement maintained.

3. Case No. 13036. A man, 24 years old. Family history of deafness. The attack commenced one year ago. Drum membrane normal. Buzzing tinnitus, and sounds like the ocean and various other noises. Politzer's acoumeter heard at 12 inches. Treatment: Astringent applications to the nasopharynx. Fifty-two days later, Politzer's acoumeter heard at 8 feet. Tinnitus very faint. The improvement has continued.

4. Case No. 13071. A woman, 74 years old. Hearing has been diminished some years. Drum membrane has small reflex and good color. It is contracted but not retracted. Congestion of promontory. Politzer's acoumeter heard in right ear at 10 feet; in left ear at 27 inches. Watch not heard. Intranasal and nasopharyngeal treatment for

thickened mucosa with stimulating astringents. Twelve days later, Politzer's acoumeter heard in right ear at 15 feet; in left ear at 42 feet. Two years later same watch heard at 4 inches in both ears. Improvement well held; very slight decrease during four years due to senile change.

CONCLUSION.

Pathology.—Exclusive of infectious conditions, there is but one fundamental pathologic condition in the middle ear which frequently interferes with normal hearing, namely, chronic interstitial otitis. All the various forms of atrophic processes, "chronic dry catarrhs" and "ostitis spongiosa of the labyrinth" come under this head. The nutritive changes may be inaugurated by local, reflex or general causes. The local causes include inflammation, infection and suppuration. The general causes work through nervous and blood supply changes.

The prognosis indicates a more or less gradual deterioration of hearing. Under rational treatment the prognosis is good for arrest of the progressive deterioration of hearing and improvement of audition in inverse ratio to the advances already made by the pathologic changes.

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XXVI.

SYMPTOMS AND DIAGNOSIS OF OTOSCLEROSIS.

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Chicago.

An insidious onset of deafness, beginning usually in early middle life, involving often but one ear primarily, yet as a rule including later both ears, associated usually with tinnitus aurium, and leading frequently after the lapse of years to profound deafness, this forms the usual clinical picture of the condition known as otosclerosis.

The gradual onset forms one of the most characteristic symptoms and on account of this insidious character it frequently happens that when the process is limited to one ear, and especially where tinnitus aurium is absent, the deafness becomes far advanced before the patient is aware that he has any defect in hearing. On the other hand in exceptional cases the progress of the defect in hearing is quite rapid, so that in the course of a few months' time the defect already becomes very marked.

There is no other form of ear trouble which so frequently involves both ears as does otosclerosis. This conforms with our knowledge of the etiology that some underlying hereditary condition is the all important factor. Still it is not uncommon for the process to be confined to one ear for a number of years, producing a profound defect in the hearing in that ear before anything can be detected on the opposite side.

Tinnitus aurium in some form is usually present. Its onset may antedate any detectable disturbance in hearing. On the other hand it may appear after the defect in hearing is well advanced. It may be intermittent, although as a rule it is constant. In pitch it may be high or low, and it is not uncommon for patients to complain of several quite distinct types of tinnitus at the same time. I have several times seen patients who described three distinct types of subjective noises with sometimes the one and at other times another temporarily increasing, so as to practically drown

out the other two. A temporary increase in the subjective noises is usually associated with an increase in the deafness.

The deafness does not by any means invariably become severe. Often the progress of the condition is either so slow that no increase in the deafness can be detected for a number of years, or else there are periods of longer or shorter duration when the condition remains practically stationary.

There are other symptoms which are complained of with more or less frequency by patients suffering from otosclerosis. These are a sensation of fullness in the ear, slight pain deep in the ear, alteration in the severity of the deafness and the subjective noises depending on the condition of the weather or the presence of physical or mental fatigue. These patients are also frequently subject to slight attacks of vertigo attributed to an involvement of that part of the labyrinthine capsule covering the semicircular canals and the vestibule. Paracousis Willisi is more marked in otosclerosis than in any other form of ear trouble. Objectively the occurrence of Schwartze's symptom is the most significant. This consists in the presence of a reddish glow transmitted to an unusually transparent membrana tympani from a congestion of the mucous membrane covering the promontory. This reddish glow may be constantly present or it may be brought out only after catheterizing the Eustachian tube.

In the diagnosis of otosclerosis this condition must be distinguished on the one hand from cases of nerve deafness, and on the other from deafness due to middle ear disease.

In the diagnosis from nerve deafness it will be convenient to consider separately three distinct types of otosclerosis. First, those cases where the defect in hearing is due entirely to the fixation of the foot-plate of the stapes; second, those cases where in addition to the defect in hearing caused by the fixation of the foot-plate of the stapes there is also a more or less extensive defect in hearing caused by an involvement of the structures in the cochlea, and third, those cases of otosclerosis where the spongifying of the capsule of the labyrinth has produced no interference with the action of the foot-plate of the stapes, and where the

defect in hearing is therefore due solely to disturbance in the function of the structures in the cochlea.

In the first group of cases, where the defect in hearing is due solely to the fixation of the foot-plate of the stapes, the diagnosis from nerve deafness presents no difficulties for even in the incipient stages of this process, and where the defect in hearing for the voice is but slight the interference with the action of the stapes already produces the prolongation of the bone conduction, the shortened positive, or even negative Rinne, and the elevation of the threshold for the perception of the lower tones which are characteristic of an obstruction in the conducting mechanism. In nerve deafness just the opposite reactions are found. Here the duration of bone conduction is more or less shortened, provided both ears are involved. The Rinne is always positive, when the tuning fork can be heard at all, except of course in those cases of nerve deafness which are limited to one ear where the Rinne will be negative, provided the defect in hearing is at all marked. In partial nerve deafness, too, the defect in hearing is limited, as a rule, to the upper part of the scale, although of course it is not impossible in nerve deafness to have a disturbance limited to the upper coils of the cochlea with a defect for the lower tones while the higher tones will still be heard normally.

In the second group of otosclerotic cases, where in addition to the defect in hearing produced by fixation of the foot-plate of the stapes, there is also a more or less extensive defect due to involvement of the structures in the cochlea, the diagnosis is often by no means so clear. This is particularly true in those advanced cases where the labyrinthine deafness is extensive, a condition to which the term disacousis has been applied.

The underlying pathologic process in otosclerosis, the spongifying of the bony capsule of the labyrinth, while beginning in most of the cases in the immediate neighborhood of the fenestra vestibuli and in this way producing usually an early fixation of the foot-plate of the stapes, is not always restricted to this part of the labyrinth capsule. The same change may take place in other parts of the capsule as well, producing as we have already pointed out the disturbance in equilibrium, when the vestibular part of the capsule is involved. In the same way, apparently, disturb-

ances in the function of the organ of Corti are produced when the adjacent capsule of the cochlea becomes involved in this spongifying process. These disturbances in the cochlea appear to be circumscribed, and limited in a measure at least to the areas in the cochlea in more or less close relation to the part of the capsule involved. It is probably in this way we have produced in most of the typical cases of otosclerosis, even in the incipient stages, a defect for the higher notes of the Galton whistle, because that part of the organ of Corti stimulated by the higher notes of the Galton whistle lies under the promontory in close relation to the fenestra vestibuli, in other words, in close relation to the part of the labyrinth capsule involved in the spongifying process. Bezold has found cases of otosclerosis with circumscribed defects in the middle of the scale for part of the Galton whistle, and I have this past year studied a case of probable otosclerosis in which was found a circumscribed tone island as well as a circumscribed defect in the midst of the tone scale.

With the knowledge of the fact that in otosclerosis the disease of the capsule of the labyrinth is not always limited to the region of the fenestra vestibuli, but may spread more or less extensively throughout the capsule of the cochlea, producing a loss of function of the part involved, we can readily understand these cases of disacousis where in addition to the defect in hearing produced by the fixation of the foot-plate of the stapes there exists a more or less extensive involvement of the capsule of the cochlea with symptoms of nerve deafness. These cases fall naturally into two groups: first, those cases where in addition to the defect in hearing produced by the fixation of the stapes there is a defect due to a more or less extensive involvement of the basal coil of the cochlea. In these cases we will find in addition to the elevation of the lower tone limit a more or less extensive defect for the higher notes of the Galton whistle, yet the Rinne will be found to be negative and there will be found a prolongation of the bone conduction.

The left ear in the following case is an example of this group. Mr. B., 54 years old. His father's mother was deaf for many years. His father had a sister 70 years of age, who was quite deaf. Of her six children two are somewhat

hard of hearing. The patient's father began to be troubled with impaired hearing at the age of 25. He lived to be 73 years old, and suffered for many years from severe deafness. The patient has one sister 44 years of age, who has been hard of hearing for fifteen years. He has one brother 50 years old, whom I have examined, who is suffering from an advanced stage of otosclerosis with more or less extensive nerve deafness, as evidenced by a marked defect in the upper part of the scale. The patient himself began to notice loss of hearing when he was 14 years old, first in the right ear, but later in both. He has always had some tinnitus aurium, much worse in previous years than lately, and Paracusis Willisi is very strongly developed. An examination found the drum membrane practically normal with slight thickening, and the Eustachian tubes normal. Whisper could not be heard in either ear. Spoken voice "28" heard somewhat clearer in left ear. In the Weber test the A fork is lateralized indistinctly to the left ear. The Schwabach test shows marked prolongation of bone conduction. Rinne for the a^1 fork is strongly negative for both ears. The lower tone limit for right ear is 30 d.v. There appears to be in this ear an indefinitely limited defect in the scale between h and c^4, and a well marked tone island from c^4 up to mark "14" on the Edelmann-Galton whistle. From "14" to upper limit there is a total defect. In the left ear the lower tone limit is 40 d.v. All tones above 40 are heard up to mark "11" on the Edelmann-Galton whistle. Above "11" there exists a total defect. In this case the defect in the lower part of the scale, that caused by the ankylosis of the stapes, is greater in the left than in the right ear, but the defect throughout the scale and at the upper limit, that caused by the involvement of the cochlea, is more extensive in the right ear. The result is that the patient relies on the left ear exclusively for hearing, and it is to this ear that the bone conduction is lateralized in the Weber test.

The second group of cases of disacousis includes those where in addition to the defect in hearing produced by the fixation of the foot-plate of the stapes and the loss of hearing for the higher tones produced by the spongifying of the promontory there exists extensive nerve deafness throughout the middle of the scale, evidently because of the

involvement of the capsule of the cochlea at a distance from the oval window. The right ear in the case just cited is an illustration of this group. The functional tests in these cases, provided the hearing in the opposite ear is gone or both ears are similiarly involved, may give all the typical reactions due to nerve deafness, that is a pronounced defect in the upper part of the scale for the Galton whistle, a shortened bone conduction, and a positive Rinne for the a^1 fork and in addition there will be the marked elevation of the lower tone limit due to the fixation of the stapes. Whether the Rinne will be positive or negative in such cases depends upon whether the deafness for the particular fork used in making the test is part of the defect in hearing resulting from the fixation of the stapes, or whether the deafness for this fork is the result of disturbance in the cochlea due to the spongifying of its bony capsule. In the first case the Rinne will be negative while in the latter it will be positive. This also applies to the results obtained in making the Schwabach test. The duration of bone conduction may be prolonged or shortened, depending on whether the defect for the fork used is caused by the fixation of the stapes or by changes in the structures in the cochlea. When these facts are kept in mind we can readily understand the explanation of such results as a prolongation of bone conduction for the A fork and at the same time a shortening of bone conduction and a positive Rinne for the a^1 fork in the same case, a result not infrequently met with.

We come now to the third general group of otosclerotic cases, those cases where the foot-plate of the stapes has remained free, and where the spongifying of the capsule of the cochlea has produced more or less extensive nerve deafness.

Siebenmann was the first to report the postmortem findings in such a case where the functional tests indicated nerve deafness, and where foci of spongifying in the capsule of the cochlea established the diagnosis of otosclerosis.

The diagnosis of these cases from the other causes of nerve deafness cannot, with our present knowledge, usually be made. In some of these cases, however, the diagnosis of otosclerosis can still be made with a reasonable degree of certainty. One such case I presented before the Chicago

Laryngological and Otological Society in 1903.[1] This case was briefly as follows: A young man, 27 years old, a university student, general health good, no previous general illness, and no history of syphilis, began three years previous to detect insidious development of tinnitus aurium and defect in hearing, first in one ear, but after a few months involving both ears. There was complete absence of any evidence of middle ear catarrh. The membrana tympani in both ears was quite normal, but showed in a most characteristic manner the reddish glow transmitted from the wall of the promontory, which Schwartze first pointed out as characteristic of otosclerosis. Here we have a history with symptoms and physical findings typical of otosclerosis yet the functional tests discovered the findings typical of an involvement of the inner ear. They were as follows: Whispered voice heard in the right ear only when close to the meatus, in the left ear at six inches from meatus. The Weber test lateralized in the left or better hearing ear. The duration for bone conduction was markedly shortened and the Rinne was positive. The a^1 fork (64 d.v.) was heard by air conduction normally in both ears, while the hearing for the c^4 fork was markedly shortened. From the history of this case with the physical findings and the results of the functional tests I believe the diagnosis of otosclerosis was justified, the spongifying process having involved extensively the basal coil and even the wall of the promontory, as shown by the presence of Schwartze's symptom, producing the typical picture of nerve deafness, but leaving, up to that time, the foot-plate of the stapes free, and therefore giving none of the results of obstruction in the conducting mechanism so characteristic of the usual case of otosclerosis.

I have studied another case this past year where the functional examination shows an extensive involvement of the cochlea, and yet where the absence of any constitutional disease that is known to produce labyrinthine deafness, and the fact that the process was an insidious one, coming on before the age of 20, when taken with the fact that a brother had developed the same type of deafness, coming on in exactly the same way, led me to believe that we had probably to deal with otosclerosis in which the basal coil of the cochlea was involved without producing any interference with the foot-plate of the stapes.[2]

The diagnosis of otosclerosis with fixation of the stapes from deafness due to middle ear disease can, as a rule, be readily made. Whenever there is found a normal drum membrane and Eustachian tube in a case of insidious development of deafness where the functional tests show an interference with the conducting mechanism, such as a prolongation of bone conduction, a negative Rinne, and a decided elevation of the lower tone limit, the diagnosis of otosclerosis is justified.

In the incipient cases of otosclerosis, where the defect in hearing is still only slightly developed, the question will sometimes arise whether this defect might not be due to an adhesive middle ear catarrh in which the alteration of the drum membrane is so slight as to be scarcely discernible. Yet in these incipient cases the diagnosis of otosclerosis can often still be made with a reasonable degree of certainty by finding the evidences of obstruction in the conducting mechanism, the prolongation of bone conduction and the elevation of the lower tone limit, developed out of proportion to the degree of deafness.

It is often claimed that a diagnosis of otosclerosis in these incipient cases can be made by noting the absence of improvement in hearing after inflation, whereas in beginning deafness due to catarrh of the middle ear, considerable improvement after inflation is usually noted. This test cannot always be relied upon, as one occasionally finds considerable improvement in hearing after inflation in these cases of beginning otosclerosis, where the foot-plate of the stapes is still apparently not firmly fixed by ankylosis. I have under observation now a young woman who consulted me a few years ago because of beginning deafness and tinnitus aurium. After making an examination a diagnosis of incipient otosclerosis was made, yet in this case, after half a dozen inflations with massage of the ossicles, the hearing improved for the whispered voice from 2 feet to 9 feet in the one ear affected at that time. The subsequent history has shown conclusively that the diagnosis of otosclerosis was correct, although the improvement in hearing continued for the greater part of a year before the progress in the condition overcame the temporary loosening up of the stapes thus brought about.

Still more difficult of diagnosis than these incipient cases

are those cases where an otosclerosis develops on top of some previous middle ear disease. Anything like a positive diagnosis in such cases is usually out of the question. Still otosclerosis may be suspected in spite of the presence of alterations in the membrana tympani where the development of the deafness is an insidious one, coming on in early life, and where the functional tests show in proportion to the degree of deafness an exaggerated prolongation of the bone conduction and negative Rinne, together with a marked elevation of the threshold of hearing for the lower tones and a well-developed defect for the higher notes of the Galton whistle. The diagnosis of otosclerosis will be all the more certain in such cases when the history shows other cases of deafness in the same family coming on insidiously in middle life and resulting finally in profound loss of hearing.

With the knowledge that the process known as otosclerosis or spongifying of the capsule of the labyrinth is by no means restricted to producing bony ankylosis of the footplate of the stapes, but that there occurs at times a more or less extensive involvement of the capsule of the cochlea, producing symptoms of nerve deafness, as well as of deafness due to fixation of the stapes, the problem of diagnosis becomes at times one of the most difficult in the whole field of otology.

On the other hand, as I have attempted to bring out in this paper, with the knowledge we now have of the pathology of this condition, the manner of onset, and the associated clinical symptoms, the diagnosis of otosclerosis not only from nerve deafness, but from other conditions producing obstruction in the conducting mechanism, can usually be made with all the positiveness which any problem in diagnosis permits.

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XXVII.

THE NON-ACOUSTIC FUNCTIONS OF THE LABYRINTH: A REVIEW OF OUR PRESENT KNOWLEDGE OF THE ORGANS OF STATIC AND DYNAMIC EQUILIBRIUM.*

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In presenting to this body a compilation and abstract of the work of others in the form of an historical review and a formulation of our present knowledge of the function of the inner ear in relation to equilibrium and orientation, I feel the requirement of justification. My plea is the subject itself, which is one of increasing interest and growing practical importance. Incomplete and hasty as the review must be in the time at my disposal, a sifting of the evidence and a summing up from the standpoint of practical otology may not be without value.

The history of systematic and scientific investigation of the physiology of the inner ear and especially of the complex function of equilibrium dates back for over a century. The questions involved are so intimately related to other chapters of physiology, notably of the visual perceptions, the sense of space, direction and position, and they are so often suggested by the practical experience of the clinic and operating room, that workers in the most varied fields have contributed to our present knowledge. We have the exact and careful animal experiments of Purkinje, Flourens, Goltz, Ewald, Breuer, and many others; the clinical obser-

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The writer has made free use of the chapters on equilibrium and functional tests in W. Nagel's Handbuch der Physiologie des Menschen, Vol. v, 1905, and Geo. Boeninghaus' Lehrbuch der Ohrenheilkunde, Berlin, 1908, many passages being cited verbatim, especially from the last named excellent treatise. The anatomic details follow Schwalbe, Anatomie der Sinnes-Organe, in Bardeleben's Handbuch, Jena, 1905, with modifications after recent writers.

vations of Lucae, Bezold, Meniére; the operative findings of Schwartze, Jansen, Hinsberg, Passow, Kipp and Burnett; the neurologic investigations of Bechterew and Baginski; the patient elaboration of tests by v. Stein, Barany, Krotoschiner, Koranyi; the studies of the functional defects of deaf-mutes by James, Bezold, Kreidl, Mygind, and the psychologic applications and analyses of Cyon, Muensterberg and Warren, while in the important field of visual equilibration we are indebted to Helmholtz, Hering, Aubert, Delage and a host of physiologists for detailed tests of normal reactions and an arrangement of the varied data on the basis of uniform and simple principles.

Within the last ten years the literature in this wide field has become so voluminous that its mere citation would be impossible. It follows of necessity that many investigations have been omitted and others barely mentioned in my review. I have endeavored, however, to do justice to the subject by paying most attention to fundamental advances both in theory and practice and to the work of those investigators whose original experiments and observations have been most valuable.

The advance of clinical otology and particularly of its surgical branch has of late been striking and gratifying. One of the most important conquests has been the systematization of operative indications and technic in the surgical diseases of the middle and inner ear and their not infrequent intracranial complications. Many theoretical questions in diagnosis and symptomatology have been answered, and many anatomic minutiae learned, by the findings at operation and by the results, negative and positive, of surgical intervention. For the purposes of the aural surgeon the possibility of the application of all symptom interpretation, and of all diagnostic methods will depend in the last analysis on the degree to which they conform with and explain the manifestations of disease in actual practice. While we must admit this without reserve, there can, I think, be no doubt of the value of continued study of function and its disturbance from the experimental theoretic side, no doubt that an elaboration and critical analysis of all rationally scientific methods will be of eventual service to the practitioner, and that the final result of accurate and painstaking investigations in laboratory and clinic will

bring theory and practice into much desired accord and correspondence. The study of the functions of the inner ear and the systematization of functional tests is by no means complete, but enough has been done to show that we are moving in the right direction. The objections which have been raised to the errors inherent in this or that test, the exceptions to minor points of this or that theory are details, not fundamental, and cannot invalidate the principles involved nor prevent their eventual application. In fact, the practical objections of competent clinicians and surgeons can only serve a good purpose in correcting errors while they stimulate anew the search for the truth.

BIOLOGIC BASIS OF EQUILIBRIUM, TROPISMS.

A study of that complex of sensations and reflexes, of inhibitions and innervations, of conscious adjustment and automatic correction which is summed up and comprised in the term equilibrium, may be facilitated by reference to the simpler conditions existing at a lower level of structural and functional development. Biology has shown that there is present, and possibly inherent, in all forms of multicellular life, a tendency to show definite, and apparently purposeful reactions, so-called somatic reactions, to a large number of cosmic influences. In contradistinction to the visceral reflexes, which have to do with the internal organs and their functional response to stimuli in the form of such acts as swallowing, breathing and so on, these so-called somatic reactions consist in changes of position or attitude and may be produced by a number of agencies. As these agencies evidently act as stimuli, and the response motion has the effect of altering the relation of the position of the organism to the source of stimulation, these changes have been grouped under the title of "tropisms," or turnings in a wider sense, and meaning not so much a rotation as a movement toward or away from the source of irritation. It has been shown, notably by Loeb and his school, that there is a uniform tendency to react to darkness and light by somatic alterations (heliotropism); to gravitation (geotropisms), to electrical current (galvanotropism), and to heat as well as to mechanically produced air vibrations (rhythmotropism). The varied changes of attitude and

position can all be reduced, teleologically, to a simple formula or principle, the gist of which is that a final optimum of position is secured for the organism. This optimum may differ for various genera, species, and even individuals, but it is generally characteristic and imposed by the needs of the particular class, and by the desiderata of equal symmetrical innervation and a maximum saving of sensory as well as motor energy.

With increased complexity of structure and a differentiation of the body form into head and trunk, upper and lower, or abdominal and dorsal aspects, the somatic reactions to gravity become more and more accurate and delicate, and finally produce characteristic positions of balance and equilibrium both at rest (static) and in motion (dynamic). In higher animals, these automatic adjustments are found to be influenced by volition and as the cerebral hemispheres develop in the ascending series of animals, phylogenetically, there is evident an ever increasing domination, through inhibition and active innervation, by the will and by intellect. The highest stage of this domination, notably in man, includes the recognition of a summation of subjective sensations of an ego, as opposed to an objective something outside produced by a systematized externalization of perceptions. This process it is which finally produces the conception-complex of ideal space or direction to which we unconsciously refer, and with which we compare our motions and sensations. Association, memory and habit combine to produce nerve paths of least resistance and to produce in sensory organs and cerebral centers so-called local signs, which may be defined briefly as definite conceptions of the relation of external agencies as cause, with subjective sensations as effect. Thus we have retinal local signs corresponding to different eye positions, and to different degrees of innervation of the ocular muscles, to accommodative states and to various points in space; cerebral local signs for all these data and for all of the varied sensory perceptions.

FACTORS OF EQUILIBRIUM.

The adjustment of the individual to space, equilibrium, and his recognition of that adjustment, orientation, is, in man at least, a complex function. Touch and sight, muscle

sense, in its widest meaning, sensations of pressure or tension, of lightness or heaviness, in tendons, joints and viscera, as well as the feelings produced by consciousness of changes or states of hyperemia and anemia, all play a part. Nagel has shown that many of these agencies act so automatically that they may not enter fully into our consciousness, and special adaptations of conditions are required to exclude one or the other in the attempt to analyze and explain a single one. In fact it has been questioned whether this function of balance is a sense at all and whether it is not rather to be considered as an unconscious judgment-reaction of the sentient organism to the sum of sensory impressions. This question is of psychologic rather than practical importance, and it has been found possible to make a fairly accurate and complete analysis of the functions of each factor. The part played by the visual organ has been studied with special ingenuity and exactness, and the work of Helmholtz, Heryng, Aubert, Fechner and many others has finally given us a clear idea of the ocular function in space perception.

Stated very briefly and generally, there is a definite and constant reaction to visual stimuli, having as its object the production of an optimum position of the eye-ball and clear central vision of external objects by means of macular fixation. This is expressed by a tendency to preserve the relation between verticality of the corneal meridian with verticality of external objects, to compensate for inclination of these objects by an opposite inclination of the cornea, and secondarily but necessarily, of the retinal meridians. This correction is automatic and unconscious, and is complete for small degrees of inclination below 10°, that is, for alterations below the limit of perception. It is rather striking that this correction fails at a point where, as we shall see, another sensory-motor mechanism is brought into play by the mechanical conditions arising with higher degrees of inclination.

Fixation of objects at rest, a second and important factor of space perception, is a summation of all the ocular motions necessary to produce clear macular vision. The position of the fixed object is unconsciously judged not only by the stimulation-sense of the muscles engaged in fixation, but by a reference to the sense of position in our own bod-

ies. There is here a definite interrelation of the subjective and objective, objects being referred to the known or felt position of the subject, and the position of the subject also judged, at least in part, by known positions, known that is from experience with objects in the external world. Fixation of moving objects also follows an instinctive tendency to bring them into the range of macular fixation. In the case of definite motions, that of objects progressing through space in one and the same direction, we can recognize two main divisions of eye motions. First, a rapid associated turning of the eyes toward the source of the moving objects, that is against their direction, real or supposed, for the purpose of catching that object on the macula. This motion, as has been shown by numerous experiments, is not associated with vision. In fact during this, recovery or compensation leap (the "Einstellung" of the Germans), there is no clear vision whatever. The second motion is in the direction of the moving object and consists in keeping step with it, as it were, so that it is always in the line of sharpest vision. As this motion is regularly and invariably associated with a definite form of motion in external objects, its production, by any means whatever, will produce a sensation of such motions. It has also been shown that our knowledge of subjective motion is largely dependent on the feeling of eye-muscle-motion which accompanies the change of position in external objects. Numerous illusions of motion are due to apparent changes in the position of external objects. We are all familiar with the false sensation of moving in an opposite direction when in a stationary railroad train past whose windows a train of cars is slowly moving. We are largely dependent on our eyes for our habitual sensations of motion and change of position, and when deprived of these, as in the dark, our sense of motion direction may be greatly diminished. This is particularly true of rotation-motions and the lying position. Progressive motion, forward or backward, in the erect position, is uniformly attended by sensations of resistance or friction between the body and its support. The absence of these tactile and visceral sensations, or their changed relations to our experience, accounts for the ease with which we can be misled as to the direction in which we are moving when lying down, as at night in a sleeper or on shipboard.

Besides the definite subjective sensations produced by changes of position, we note definite somatic reactions, "specialized tropisms," we might call them, having as their conscious or automatic object the restoration of balance, i. e., the regaining of the optimum position, which we may assume is normally and habitually occupied. This adjustment is in the nature of a correction or compensation. It depends upon muscle motions, largely involuntary and reflex, brought into action by sensations of lack or loss of balance. It is significant for the safety of the organism that disturbances of the motor arc of this mechanism do not vitally affect or seriously interfere with balance. Differences of muscle strength for instance, in the two sides, even marked irregularities, such as a flat foot or a sprained ankle, do not cause loss of equilibrium. There is probably unconscious adjustment for these factors, as there is for the unequal strength of right and left hand when both are used together in pulling or pushing. The sensory component, on the other hand, is of the greatest importance, as it is through the sensations of resistance that the varying degrees of muscle innervation are called into action, and as a matter of fact, disturbances of sensation, anesthesias, are particularly apt to cause confusion in somatic reaction for the preservation of equilibrium.

THEORIES OF THE FUNCTIONS OF THE LABYRINTH.

The striking anatomic peculiarities of the structures of the inner ear, the snail-shell whorls of the cochlea, the regular curves and geometric arrangement of the semicircular canals, and the suggestion of complex function and of necessary purpose afforded not only by structure but by their presence throughout a long range of the animal kingdom, attracted early attention and inspired investigation, while they seemed to hold, as it were, within themselves the answer to the riddle of their meaning. The arrangement of the canals, particularly, in three principal dimensions of space was at once felt to be of fundamental significance, and from the earliest years of modern scientific investigation we find this special relation as the starting point of theory. The most obvious connection was that with audition, and it was assumed that the three semicircular

canals mediated sensations of sound coming in any of the three main directions corresponding to their axis, or analyzed atypical sound directions by proportional representation in the three dimensions. This theory crops up at intervals in the history of the investigation and has persisted until the present time. It is associated with the names of Autenrieth (1802), Cyon (1854), Brueckner (1888), Hensen (1893). Many variations were played on this theme. The function was thought to consist in a destruction or dampening of tone-waves which had already acted on the auditory organs, sensu strictiori (Czermak, 1860; Malinin, 1866), in perception of noises as opposed to tones, i. e., non-periodic vibrations (Schiff, 1857; Hasse, 1867); in perception of muscle tones produced by contraction of head muscles and thus indirectly of position of the head in space (Bornhardt, 1872); by bone conduction of external sounds (Ogston, 1869), and with the purpose of directing eyes and ear toward the source of sound (Münsterberg, 1889). A teleologic modification of this basic theory allowed the inference that a special sense of direction, acoustic stereognosis, as well as the homing instinct of animals was mediated by this appartus. Among other theories which attribute a non-acoustic function to the labyrinth we may mention those of Cyon and of Högyes, who believed that the nerve terminals in the labyrinth were intended to produce definite tetanic positions of the eye muscles, the 2x6 nerve endings being in communication with the 2x6 eye muscles for the purpose of producing unconscious eye positions which are not compensatory but reflex.

NON-ACOUSTIC THEORY (BREUER, MACH, CRUM BROWN).

The theory of a specific, equilibristic function of the labyrinth was based almost from the beginning on the results of careful experiments on animals and human beings, and accordingly progressed by slow and steady steps, rather than by leaps and bounds. Among the earliest observations were those of Purkinje (1820), on rotation and vertigo, and those of Flourens (1825) on experimental section of the horizontal semicircular canals. The latter investigator found in doves and rabbits that hearing was not affected, but that there was a typical disturbance of equilibrium, with characteristic

compensatory movements of the head and body. Section of the other canals caused similar pendulum motions of the head in the axis of the injured canal. Nearly fifty years later, Fr. Goltz, in Strassburg, took up the forgotten experiments of Flourens and repeated and extended them, showing that the compensatory motions were not forced motions and did not depend on auditory sensations, but were specific sensory reactions. The semicircular canals are the sense-organ for the equilibrium of the head and mediately for that of the entire body. The adequate stimulus is the gravitational pressure of the column of endolymph. A few years later, Breuer and Mach, in Germany, and Crum Brown, in England, suggested a modification of the theory of Goltz. The first named author called attention to the fact that the mechanical requirements of this theory could only be satisfied if the semicircular canals were suspended in a medium like air, of a specific gravity appreciably lighter than that of the endolymph, whereas, in fact, they are surrounded by perilymph which is of about the same density. The modification of Breuer and Mach consisted in the hypothesis of endolymph motion in place of endolymph weight-stress as an adequate stimulus, and this slight change was the cornerstone of a new structure. As the theory of Breuer and Mach with its adaptations to advances in experimental and clinical investigation is the most complete and satisfactory at the present time, and as most of the clinical tests and diagnostic conclusions are based on acceptance of its fundamental assumptions, somewhat more detailed consideration of this theory may be advisable.

PLAN OF SEMICIRCULAR CANAL SYSTEM.

I shall not tax your patience with a description of the anatomy of the labyrinth further than to call attention to the fact that we have to deal with a bilateral, symmetrical structure. Each canal of a side corresponds in plane to a canal of the opposite side; thus the two external canals in the horizontal plane, the anterior or superior canal of one side with the posterior or inferior of the other side. (Fig. 1.) It will also be noticed that the ampullar bulbs of corresponding canals are at opposite ends; the ampulla of the right horizontal being on the right, while that of the contra-

lateral canal is on the left; the ampulla of the right superior canal being in front, above, and that of its synergist, the left inferior, being behind, below. Furthermore, the greater part of each canal is posterior to the ampulla, so that bearing in mind that the entire system is about on a level antero-posteriorly with the axis of rotation of the head, any rotational change will correspond to an opposite rotation of the corresponding canal. The practical significance of this arrangement will be pointed out in the analysis of the functional mechanism of the labyrinth.

ANATOMY OF THE LABYRINTH.

The membranous labyrinth (Fig. 2) containing the terminals of the vestibular branch of the acoustic nerve is enclosed in a bony capsule, the osseous labyrinth, occupying the middle of the petrous portion of the temporal bone. In animals of the higher vertebrate class the structure is very like that in man. The sagittal axis of the system is directed inward at an angle of about 45°, and the horizontal canal is not in a horizontal plane, but inclined backward at about the same angle.* The posterior crura of the two vertical canals on either side join and enter the utriculus by a common port, so that there are only five openings instead of six. Each canal has a spherical enlargement, the ampulla, at one end. Within this ampulla we find a prominence so marked as almost to occupy the central axis of the canal. On this crista we find sensory epithelium with long pliable hairs, not floating free in the endolyymph, being held together by a gelatinous mass, the cupula terminalis. At two points in the membranous labyrinth we find, respectively, a vertical and a horizontal collection of sensory epithelium, very similar in arrangement and structure to that of the ampullae, the macula utriculi and macula sacculi. The hairs are shorter and stiffer than those of the ampullae, are held together by a stiffer mass (otoconium), which, however is not gelatinous, but composed of the so-called otoliths ("statoliths" according to Verworn), microscopic crys-

*This special arrangement of the so-called horizontal canal and of the maculae would indicate that the erect position of man has not existed long enough to influence these structures of the inner ear. They are still in the same position as those of the "beasts of the field which creep upon the earth."

tals of carbonate of calcium. The rest of the wall of the membranous labyrinth has no nerve terminals, but gelatinous threads have recently been traced by Breuer from the wall of the utriculus to a drop-like accumulation of the same material on the otolith mass which they evidently nourish and renew. The plane of the macula utriculi corresponds to that of the external semicircular canal, i. e., in the upright position it is inclined backward at about 45°. The otolith plate of the sacculus, under similar conditions, runs at about the same angle from above and behind, forward and downward, in the anterior posterior plane, i. e., parallel on either side. In lower animals there is a third macula, the lagena, allowing vertical displacement of the otolith plate, while both utricular and saccular macula are horizontal, but at right angles.

NERVE PATHS AND CEREBRAL CONTROL.

The vestibular nerve (Fig. 3) starts from the ampullae, one branch (ramus medius N. viii) from the posterior ampulla going separately through the foramen singulare near the meatus internus, another from the macula sacculi, superior ampulla, and the external ampulla (first branch of ramus superior), through the upper portion of the terminal plate, and a third, utricular branch (second branch of ramus superior), through its lower portion, through the vestibular, bipolar ganglion, which according to Hensen is the neurologic nerve terminal of the vestibular branch. The vestibular nerve now enters the pons and reaches the dorsal nucleus of the eighth nerve (Deiter's and Bechterew's nuclei). Here the peripheral neuron ends. The central filaments can be traced across around the fourth ventricle and the tegmentum or roof to the tegmental nucleus, most of the fibers going to the opposite side. This path brings the vestibular nerve into relation with the anterior columns of the cord and with the abducens. There is no known connection with the cerebral cortex. The cerebellar cortex is intelligible as an indicator of degree of tonus, contraction and effort, in various body muscles, notably those of eye and lower extremities, established voluntarily by the brain, or by reflex from tactile, muscle, visual and balance sense, to preserve body equilibrium and possibly reflex fixation

of external motion. In time it would become, as we know of the cerebral cortex, a store house for memory-pictures of sensation-states connected with eye-motion, tactile impressions and muscle tonus, in their relation to balance and the perception of motion, direction, rotation and the conception of ideal space, and would thus aid in regulating subjective sensations by referring them to a common standard of tradition, experience or memory. A discrepancy of signals received at a given time, whether from eye, tactile or muscle sense, or balance centers in labyrinth, either with each other or with memory standards, would produce the effect of a contrary innervation and the markedly unpleasant subjective sensation of confusion which we call dizziness. The unconscious judgment factor in complex sensation of balance and its disturbance in dizziness are shown by the gradual development and increase during life of the capacity for both, the susceptibility of highly organized and trained intellects to dizziness, the comparative immunity of children and stolid, phlegmatic, illiterates, and the ease with which the sensation of disturbed balance or dizziness can be elicited by slight modifications and confusions of visual impressions. This is particularly the case with the swinging mirror which produces almost immediately a feeling of marked vertigo and even nausea.

The labyrinth is in connection with the ocular muscles presiding over abduction to the same side, i. e., external rectus of the corresponding eye and internal rectus of the opposite eye; the occipital muscles and trunk muscles of the same side and with lateral rotators of the head to the opposite side (Marikovszky, 1904, rabbits). The connection with the oculomotor nuclei and the anterior horns of the cord runs through Deiter's nucleus (Adler), and the central end station for centripetal sensory stimuli is situated in the cerebellum. According to Stefani, the cerebellum is a central organ whose activity is principally stimulated and maintained by impulses emanating from the labyrinth and whose function consists in the preservation of muscular tonus which is required for the maintenance of a stable body position in normal equilibrium. The cerebellar cortex is not to be considered as the center for sensations of motion and position (Lange, Nagel). The labyrinth, besides its function in maintaining tonus, mediates these sen-

sations and it is to be assumed that they have a representation in some cortical area, but this area has not yet been determined.

THE MECHANICS OF LABYRINTHINE FUNCTION ACCORDING TO BREUER AND MACH.

(Figure 4.)

With every rotation of the head the endolymph contents in the semicircular canals lags behind on account of inertia, so that in the canal corresponding to the plane of rotation there is a contrary current or pressure stress of lymph, which will produce torsion on the terminal plate and the cilia of the sensory epithelium projecting into the lumen from the ampulla, and, it is to be assumed, cause a specific irritation of the nerve terminals. The axis of head-rotation will determine in which associated pair of canals this endolymph motion will occur. In motions which are not exactly in the axis of any pair of canals, there will be an associated action of several pairs. It is evident that the physical conditions for a contrary current due to inertia are satisfied only as long as there is an acceleration of rotation of a whole canal. After uniform rotation has been instituted there can be no relative motion or disturbance of balance between contents, endolymph and canal wall. Every positive or negative acceleration, however, and particularly a sudden cessation of rotation, must produce a marked displacement of the column of endolymph, which will continue to move in the direction of previous rotation (remnant of motion). The only serious objection to this hypothesis is the extremely small caliber of the canals, which in man are only 0.1 mm. square. This has been met by the substitution, for actual motion of the endolymph, which would probably be neutralized by friction against so narrow a wall, of a pressure state in the lymph such as might produce distortion in a vertical membrane hung up in its course. The crista projecting into the lumen of the canal would represent such a membrane. (Fig. 5.)

Breuer's theory of the static function of the labyrinth, as applied to sensations of position, is based on the assumption that the otolith mass on the macula is of so much greater density than the endolymph that various head positions

must produce various degress of pressure or stretching in the sensory hairs. With a macula in the horizontal position the macula would press directly on the hairs which carry it. In the reversed position, with the otolith plate below, there would be traction on the hairs. In any intermediate position of the system there must be distortion of the hairs which would reach a maximum, say, when the plane of the otolith plate and that of the macula were vertical in space. The obvious and striking difference of this mechanism from that of the ampullae and canals lies in a persistence of the mechanical conditions and of the torsional stimulus induced by them, as long as the position of the system in space is unchanged, while the mechanical influence on the cupula in the ampulla takes place only when there is a change in the rate of motion of the labyrinthine contents. This duplicity of function, or rather of excitation modus, will serve to explain the distinction between those sensations and reflexes which are associated with motions, passive or active, of the head, and those which are due to permanent positions of the head in space.

Experiments on lower animals in regard to the function of the otoliths or otocysts, which can be removed without injury to other parts much more easily than the semicircular canals, show that they are essential for the maintenance of equilibrium. Crabs, squids, marine snails and other animals deprived of these organs, swim on one side or on the back, or in circles. These experiments with defects are supplemented by one very instructive series of irritation tests. Certain crabs shed their sand otoliths at definite periods. Kreidl let crabs renew their otoliths with iron filings and then found that the approach of a magnet from one side was followed by a tilting of the animals to the opposite side, evidently for the purpose of restoring the balance which in sensation at least, had been disturbed by the displacement of the magnetic otolith.

The position of both horizontal (utriculus) and vertical (sacculus) otolith plate is quite logically explained to serve in erect and in supine position, respectively, the former in a state of functional equilibrium when the body is erect, the second, under stress, and vice versa.

Otoliths produce sensation of position, requiring steady and prolonged strain of gravity, and are not easily set in

action by the usual degrees of motion-acceleration to which we are subjected. They would be useless if they reacted to every such impulse, as we should have subjective sensations of change of balance in every progressive motion. A forward jump would produce torsion of horizontal plate corresponding to falling backward. As a matter of fact such sensations occur only when rotational motions are combined with action of gravity, illusion as to real vertical inclination of body, and judgment of slant in surrounding objects corresponding to the resultant of gravitational and centrifugal factors..

There are certain positional reflexes in distinction to rotational reflexes, the most important of which are the compensatory positions of the eyes which occur when the head is tilted and which persist as long as this head position is maintained. A similar reaction is noted in birds and reptiles which keep the head in a certain position, in relation to the vertical, no matter how the body is pointed. These reflexes are explained by the function of the otoliths and their dependence on gravitation. As a certain deviation from the vertical is required before gravitation can produce torsion on the sensory hairs of the otoliths, we can now understand the function, mentioned above, of the retinal meridians and of compensatory inclination in slight deviations. With an increased angle of tilt, the otoliths are affected and the change in head position becomes apparent to consciousness. Tests of tilting at various angles support this view. When titled backward gradually, we underestimate the angle until 50° or so has been reached. From there on, there is an increase in the subjective sensation of tilting so that with a backward inclination of 75° we think we are in a horizontal position. From this point on the sensation of tilting increases rapidly, and when tilted so that the head points downward 15° we have the feeling of being inclined 45°, and think we are standing on our head when it is in reality only 30° below the horizontal.

THE EXPLANATION OF SENSORY PHENOMENA AND SOMATIC REACTIONS ON THE BASIS OF THE BREUER-MACH THEORY.

(Figure 6.)

The Phenomena of Rotation.—At the beginning of a rotation motion, and with every change in its acceleration there

is a definite sensation which is correctly interpreted, but this sensation disappears as soon as the rotation has become uniform (unless a change is produced by alteration of the position of the head). Retardation of the motion is interpreted by sensation as a motion in the opposite direction, and this is noted even after all motion has ceased. This subjective sensation of false motion, Mach's rotational vertigo, is explained by the motion-remnant in the endolymph, which is now active in an opposite direction to that caused while the system was in actual motion. The sudden sensation of motion coming on during uniform rotation, on turning the head actively through 90°, is also explained by a temporary acceleration imparted to the endolymph.

Reactions similar to those observed on passive rotation have been noted after irritation of a semicircular canal in which motion in the endolymph was produced by stroking or some similar mechanism (Ewald). There is invariably a turning of the head and a contrary motion of the eyes in the axis of the irritated canal (irritation test). Similar reactions were noted in ear operations when a semicircular canal was exposed (Jansen). In doves, the head was turned to the sound side if the fluid was displaced toward the ampulla, and the irritation reaction was more marked than when the fluid moved in the opposite direction, i. e., from the ampulla, when the head turning was toward the irritated side. We noted above that the ampullae are at opposite ends of each pair of canals. Bringing this into connection with the data gained by animal experiments we can understand that rotation to the right, for example, will produce a sensation of turning in this direction, the endolymph moving in the right horizontal canal from left to right toward the ampulla, as the occiput and with it the backward arched semicircular canals move from right to left. In the left semicircular canal the movement of the endolymph, in the same direction, i. e., from left to right, will be away from the ampulla and produce a minimum of irritation and, accordingly, of sensation. In left rotation we shall have a corresponding predominance of sensation in the left horizontal canal. If both sides are functionating normally there will be a correct interpretation of motion, and uniform somatic reaction on turning in either direction. If, on the other hand, there is an increased sensibility in one, this will

be associated with intensified reaction. Again, destruction of function will allow the normally subordinate reaction from the other side, to which we have just referred, to become evident, and the weak sensation and reaction from the contralateral canal, produced by motion of endolymph away from the ampulla, will produce a positive reaction. As we shall see later, this has been made use of for a separate test of the labyrinthine function of the two sides.

DESCRIPTION OF NYSTAGMUS MODEL.

(Figure 7.)

The quadrant is made of thin tin through which nails are driven at regular intervals to represent objects fixed during rotation. The nails are covered with thin rubber tubing to deaden the shock and noise, and a paper indicator stuck in the tube to show from a distance. A piece of rubber tubing is slipped over the pointer, too, to allow variation and adjustment of length, as in the "abducted" position, it does not project over the edge of the turn table as far as in normal position, and so fails to strike the nails on the quadrant.

When properly adjusted, with pointer normal, each rotation alteration of the turn table produces a deflection of the pointer, which is caught and carried backward by the peg moving in the opposite direction. When the pointer has been carried along for a space, it slips from the nail, or is released from fixation and jumps to the next object, or nail, with a rapid motion which is in the direction of actual rotation, i. e., toward the source of apparently or actually moving objects. This adjustment, or rapid correction leap, the "Einstellung" of the Germans, is the more marked of the two motions in nystagmus, and it is according to the direction of this "rapid" clonic motion that the nystagmus is characterized as right, left, and so on. It has been shown that there is no clear vision during this rapid compensation or adjustment motion, and this may be seen quite clearly when we read lines of print. There is an adjustment after reaching the end of each line, the eyes jumping back in order to fix the first word at the beginning of the next line, far to the left. During this leap we do not see a single word clearly, and only one or two letters are picked up dimly.

Rotation of the disc in either direction produces analogous results, i. e., nystagmus toward the direction of rotation. The effect of abduction, as by active fixation to one side, during rotation, can also be demonstrated on this model. If we attach a third elastic band (el_2) to the pointer so that it no longer points out radially from the center of the turntable, but, as shown by the dotted lines, is inclined toward one end of the line of nails on the quadrant, we find that the catching and release of the pointer, which as explained above corresponds to fixation-tonus and compensation-leap, are very slight when the pointer represents an eye looking backward, i. e., away from the direction of rotation, and very marked when the eye looks forward in the direction of turning. This is not only in harmony with the observations of every day life and clinical study, but can be very simply explained mechanically and visually. On looking backward away from the source of motion there is less apparent motion; the object moves through a smaller arc in the given time. If we look directly backward, for instance, there will be practically no motion, the object merely becoming "small by degrees and beautifully less." On looking forward in rotation the objects come into the field of vision sooner and are in the field of fixation longer; there is a corresponding sense of greater motion and a greater actual leap of compensation or clonic fixation; greater evident nystagmus. Mechanically we must remember that in looking forward in right rotation for instance, we consciously innervate the muscles for right abduction, so that this volitional stimulus would be added to the unconscious or automatic adjustment of fixation, produced by motion of external objects, which, as we have seen, also corresponds to abduction in the direction of rotation. In looking away from the rotation to the left, we innervate an opposite set of muscles consciously, to those which are being set in action automatically, and the two impulses are not summated as before, but tend to neutralize each other. Clinically it has been noted that nystagmus is increased on looking toward the diseased side, i. e., in the direction of apparent rotation, and that it may be apparent only in forced abduction to this side. Conversely, nystagmus may be inhibited by looking toward the sound side. This abduction also tends to inhibit or entirely prevent vertigo, and is made use of

for this purpose by ballet dancers and skaters to prevent dizziness in whirling. The head and eyes are turned backward, away from the direction as far as possible, and forced fixation in opposite abduction used to keep the eyes in line with one point, usually the center of the auditorium. In this way the motion of the head and eyes is artificially slowed and reduced to a minimum. After the body has moved through a large arc and made almost a complete revolution, the head is, actively, turned to catch up with it, and fixation to one side again takes place. In this way we have an interrupted motion of the head and eyes, giving the semicircular canals a chance to come to rest frequently and producing a small repeated reaction with which the sense of balance can cope without sensation of dizziness, in preference to allowing an accumulation of irritation through prolonged endolymph rotation, which on cessation of whirling, might produce extreme giddiness and loss of balance.

TESTS OF THE STATIC FUNCTION.

It has already been stated that the adequate stimulus for the ampullary organs is the motion of the endolymph produced by rotation. As in other sensory organs, a number of other stimuli can be made to replace the normal and physiologic irritation. Thus, condensation or rarefaction of air in the external auditory canal, especially when the external horizontal canal is exposed, will produce a change in the intracanalicular lymph stress. Even concentrated light under these conditions, will act as an irritant (Lucae). Mechanical pressure by a probe or by granulations, on the oval window will act similiarly, and thermic irritation by hot and cold water or galvanic currents, will supply sufficient stimuli. In all these cases we have definite sensations of rotation motion, and definite somatic reactions of eyes and head. The manifold and somewhat complicated phenomena may be most simply summed up as follows. Each irritation of a semicircular canal corresponds to a sensation of motion which is interpreted as taking place in the direction of the endolymph stream, i.e., as a rotation toward the irritated side. The somatic reaction consists in an attempt to fix objects in motion coming from the irritated side, as they naturally would if there were actual motion. There will be a rapid turning of eyes to the irritated side, "Einstellung,"

with a slower fixation tonus in the direction of the moving objects, i. e., nystagmus to the irritated side. The head will turn against the fancied motion in an attempt to correct the subjective sensation of displacement, and the trunk muscles on the irritated side will receive a stronger innervation or an increased tonus for the same purpose, i. e., to correct fancied motion change or compensate for apparent alteration of balance. We shall now consider the various methods of stimulating the ampullar apparatus and note the underlying principle which will explain the unifomity of reaction.

Dizziness and quite possibly nystagmus (although this has not been studied) may be caused by irritation of semicircular canals by molecular vibration of endolymph due to sound waves. The tone vibration must be of unusual amplitude to cause this phenomenon, and accordingly we note unpleasant subjective sensation of dizziness with unusually low and strong notes, as the deep contra-bass of an organ, a low pitched fog horn or the hum of a locomotive exhaust.

Physiologists have not decided whether the disturbances of equilibrium noted after destruction of the labyrinth are due to this destruction directly, say by irritation of the nerve trunk, or whether these disturbances are the expression of a predominance of the contralateral sound labyrinth and a consequent alteration of the balance of sensory perceptions in the static central apparatus situated in the cerebellum. In other words, whether we have to deal primarily wth phenomena of irritation or of paralysis, loss of function. Clinically this is not of practical importance as in labyrinthine disease we have almost always an extended period of irritation due to inflammation or hemorrhage. In patients we shall have to distinguish a stage of irritation and a stage of function loss which is secondary, i. e., develops later. A sharp differentiation is not always possible as loss of function may begin while irritation persists, just as in affections of the cochlear branch there may be loss of hearing with subjective tinnitus.

The main symptoms of irritation are nystagmus, disturbance of equilibrium and vertigo, with nausea and vomiting. Boenninghaus claims that the first three symptoms are related, inasmuch as nystagmus causes vertigo or false sense of rotation, and that this in turn produces loss of equilibrium. Nystagmus, involuntary eye motion, produces a sensation

of objective motion when the eyes are open or of subjective motion when they are closed; pseudo-rotation or vertigo as opposed to vertigo or dizziness in the wider sense such as visual vertigo, and so forth. The dependence of rotational vertigo on nystagmus has been demonstrated by Barany who showed that vertigo does not occur if the nystagmus is suppressed, e. g., after rotation with closed eyes, if the gaze is directed away from the direction of rotation. The direction of apparent rotation is in the direction of nystagmus, but the body, to compensate for the supposed displacement, deviates in the opposite direction. This involuntary compensatory reaction disturbs the balance of the body and leads to swaying, reeling, stumbling or falling to one side or the other.

Function loss symptoms consist in disturbances of balance without vertigo, and nystagmus without nausea and vomiting. They are not markedly different from the analogous irritative symptoms except that the latter are more marked, varied and irregular in intensity, in accordance with the varying degree of irritation.

In tests of the static function, nystagmus and balance disturbance offer objective evidence and are therefore more valuable than the subjective sensation of vertigo, which furthermore may be complicated with disturbances of general consciousness or indeterminate psychical reactions. Nystagmus is particularly valuable as a symptom (Barany) because it is absolutely independent of physical control.

CLINICAL TESTS FOR NYSTAGMUS.

Labyrinthine Nystagmus.—Irritation of a single semicircular canal in animals causes a definite motion of the eyes. If the irritation continues, a clonic spasm takes the place of the single muscular contraction. After destruction of the labyrinth there is neither nystagmus nor vertigo on rotation. A similar condition is noted in deaf-mutes with loss of both labyrinths. This form of nystagmus can be distinguished from nystagmus due to amblyopia. The latter is even, tremor-like, the former irregular, clonic, like the twitching motion seen in paresis of the external rectus muscle when an attempt is made to fix in abduction. In the usual erect position with the head held vertically, there is a horizontal or circular motion of both eyes to the right or left.

The two motions are not equally rapid, and the direction of the rapid motion determines the characterization of the nystagmus, as nystagmus to the right, left, and so on. The nystagmus is increased by looking in the "rapid direction." This can be explained mechanically by the fact that voluntary innervation now assists the subconscious innervation action of the muscles turning the eye in the direction of the gaze which is identical with the direction of the nystagmus. It is also possible that a greater arc of motion is offered for the compensatory effort as well as for the steady fixation component and that the sensation of external motion is more extended when the eyes are turned toward the source of apparently moving objects. This is undoubtedly the case in the reflex nystagmus which occurs when we fix objects from a moving train or a caroussel. The apparent motion of these objects and the visible nystagmus are both much less marked when we look back along the line and away from the direction of turning. Labyrinthine nystagmus may be so slight as to appear only in forced abduction in the "rapid" direction, i. e., toward the irritated side. The excursions may be large or small, rapid or slow. At times they can be seen only with good illumination or with a magnifying glass, on close inspection while the patient fixes an object held far to one side. Labyrinthine nystagmus may be elicited by a number of stimuli or it may be spontaneous. It is then a symptom of labyrinthine irritation, and is accordingly directed toward the diseased side. It may be assited by holding the head low and thus causing labyrinthine hyperemia. In labyrinthine irritation, nystagmus may be caused by simple turning of the head, which is quite unusual in health.

NYSTAGMUS AFTER BODY TURNING. (ROTATIONAL NYSTAGMUS.)

Active or passive rotation of the body about its longitudinal axis causes nystagmus in the direction of turning (Purkinje, 1825). Sudden cessation of rotatory motion is followed by reversal of the direction of the nystagmus, after-nystagmus. In labyrinthine irritation, rotation toward the diseased side may produce nystagmus after only one or two turns which also lasts longer than nystagmus following rotation in the opposite direction. The semicircular canal around which we turn is always irritated most

actively, as under these conditions the motion of endolymph is toward the terminal nerves in the ampulla. Unilateral loss of labyrinthine function does not produce any lasting alteration of the nystagmus, but it disappears entirely.

Barany has also noted that when there is a fistula in the bony wall of the labyrinth, compression or rarefaction of the air in the external meatus will produce typical labyrinthine nystagmus and vertigo. The details of this reaction have not yet been worked out.

When both labyrinths have been destroyed, as in deaf-mutes, if only one semicircular canal is destroyed, nystagmus, will, for a time, be more to the sound side. Practically there are technical difficulties in the way of accurate observation of rotational nystagmus. It has been suggested that the observer seat himself on the turn table with the patient and palpate the latter's closed eyes with the finger tips to determine the character and direction of the nystagmus. Practically it is more simple to test the after-nystagmus which takes place on cessation of rotation. In normal individuals about ten rotations are needed to produce decided nystagmus, while in labyrinthine irritation two or three turns suffice. The subjective sensation of rotational vertigo and the disturbance of equilibrium associated with it can best be tested by having the patient get up and try to walk in a straight line after rotation has ceased.

Caloric Nystagmus.—The specific action of heat and cold was discovered in animals by Baginski in 1881, and studied and applied to human beings by Barany, 1906, who first recognized its physiologic and diagnostic significance. If the ear be irrigated with water above or below the body temperature, nystagmus is produced which is more marked as the temperature contrast increases, and ceases when water of an indifferent temperature is used. Hot water acts as an irritant, i. e., as a stimulus corresponding to rotation to the tested side, while cold water acts as a depressant, with a diminution of sensation of rotation toward the tested side, or what amounts to the same thing if the body is at rest, to a motion toward the non-tested side. Accordingly hot water nystagmus is directed to the tested side, cold water nystagmus to the non-tested side. After destruction of the labyrinth there is a loss of caloric reaction. This has been proven by tests on animals (Kubo, 1906), and

observations on patients with known destruction of the labyrinth. Barany lays great stress on this point, and claims that we have in the caloric method an accurate means of determining the presence or absence of function in either labyrinth. A negative finding would mean that any objective reaction, such as spontaneous nystagmus, must be due to the labyrinth of the opposite side or to non-labyrinthine factors, such as involvement of the cerebellum, and the same author claims that the diagnosis of cerebellar abscess can be facilitated by means of this procedure. The mechanic explanation of caloric nystagmus is found (Fig. 8) in the production of currents in the semicircular canal due to the change in density of the endolymph which follows the change in temperature. Barany says: "Imagine a closed vessel filled with water at 37° C. to represent the labyrinth. Syringe one side of this cup with cold water. The water nearest this side will be cooled and thereby get a greater specific weight, tending to move toward the bottom of the cup, while on the opposite side the warm water rises. In the same way and for the same reason a movement of the endolymph is produced in the semicircular canal."

The diagram (Fig. 8) also explains sensory and motor reactions to rotation. a + shows effect of positive irritation of R. ampulla, by rotation to right, accompanied by homonymous nystagmus (R) and compensatory head turning (L). The canal rotates from left to right, producing a stress toward the ampulla (serpentine arrow at B.), and sensation of motion to R. b — shows negative or reversed stimulation, of ampulla, immediately after cessation of rotation, producing reverse stress in endolymph, sensation of reversed motion, and movement of objects to opposite side. Accompanied by after-nystagmus, heteronymous (L.) corresponding to sensation of subjective and objective motion. Compensatory head turning and body motion, as before, opposed to direction of nystagmus, are (R.) to restore balance, supposedly disturbed by rotation to left. This after-nystagmus and after-vertigo are easily observed after a few rotations when the subject attempts to stand and walk straight forward. It is also frequently seen in every day life, after whirling in dancing or skating. The dizziness does not come on until the rotation has ceased, but then takes place immediately, and the subject reels

in the direction of previous rotation, after coming to a stand still, as if by some inward momentum. In fact, it is due to an attempt to correct the supposed disturbance of equilibrium or position. The vertigo may be inhibited, partially or wholly, by forced fixation away from the direction of motion, real or apparent. b — also shows the effect on the R. ampulla of turning to L. during passive or active rotation. The endolymph motion (serpentine arrow) is away from the R. ampulla, hence weak, and may be described as producing a weak sensation, of turning away from the right, and corresponding in every particular excepting intensity to the sensation reactions on right rotation in the left external ampulla. It is conceivable that after loss of function in the left labyrinth, this ordinarily subordinate sensation might be sufficient to cause an independent reaction and gradually compensate, in part, for the lost function of the contralateral labyrinth.

Nystagmus might be viewed as part of the somatic reaction having for its object the establishment of an optimum position of the eyes, corresponding with macular fixation of an external object coming into the field of vision from the source of apparent motion, or with corresponding subjective change of position; in other words, an equalization of visual stimuli, and as an aid to balance to be gained from vision by fastening on, and as it were being supported by a sense impression based on objective data. The nystagmus observed in disease and in experiments must be considered as running in habitual channels. The purposive element, of course, no longer appears, but that element can be detected and is logically to be explained. Thus there is no actual attempt at fixation in labyrinthine disease or rotational nystagmus, as is shown by persistence of this ocular motion when the eyes are closed or exclusion glasses worn, but the memory of this tendency, the local sign and tradition of this intuitive reaction persists and comes out, nonpurposively as a symptom.

TECHNIC OF CALORIC TEST.

To produce an even stream of long duration and what is important in labyrinthine suppuration, as little mechanical irritation through pressure of the stream as possible, it is not advisable to use a syringe. A large Politzer bag (Boenninghaus) or a fountain syringe hung a little higher than

the ear may be used. A fine ear irrigating tip or an elastic middle ear catheter, introduced thorough an aural speculum is then carried close to the drum, an assistant managing the Politzer bag while the surgeon observes the eye-motions. These generally appear in from three-fourths to one and three-fourths minutes with intact drum, but in case of perforation they may be seen with good illumination in a few seconds, and last for several minutes. Cold water, at a temperature of about 50° to 60°, i. e., 40° lower than body temperature, is best for practical reasons as a like contrast could not safely be produced with warm water as the temperature would have to be over 130°.

Galvanic Nystagmus.—Breuer showed (1888, 1903) that on galvanic irritation of the exposed semicircular canal of the dove, head and eye movements took place, and that this reaction could be prevented by cocainization of the corresponding ampulla. After bilateral destruction of the labyrinth there is no reaction to galvanism. Similar conditions were noted in deaf mutes with total absence of labyrinth (Pollak, 1893). These observations exclude the possibility of cerebellar involvement in the galvanic reaction. The cathode acts like hot water, i. e., as an irritation corresponding to rotation toward the tested side. The nystagmus, as before, is toward the same side, and head motion to the opposite side. This appears to show that the galvanic current, running as it does from cathode, negative pole, to anode, positive pole, acts on the ampullar nerve terminals exactly as an endolymph current would when moving in the same direction, so that, translated into terms of subjective sensation, the passing of a current through, say the right horizontal canal from right to left would be felt as an endolymph motion would, i. e., as a turning motion to the right or irritated side. The anode causes the opposite reaction and is analogous to that of cold water, nystagmus to the "sound" side, and head turning to the side of irritation. Barany places the anode on the ear to be tested, and the cathode in the opposite hand. This is preferable to the transverse current through the head, as it allows either labyrinth to be tested separately. Nystagmus occurs more easily when the gaze is directed toward the cathode, i. e., again toward the source of apparent motion and of actual irritation, and toward the direction of subjective ro-

tation. It can be elicited by currents of 2-6 milliamperes; while with the gaze in the opposite direction, 10-15 are usually required. Barany has noted that in labyrinthine destruction it is impossible to elicit galvanic reaction from the destroyed side, a complete analogy with caloric nystagmus. Babinski (1901) attached more importance to the compensatory head turning, which he found always took place toward the diseased side, when this side was stimulated either from cathode or anode. This "ear phenomenon" of Baginski is only found in unilateral disease of the static apparatus, according to Mann (1907); a significant observation requiring further study. Deaf mutes without rotational vertigo also show a loss of the galvanic reaction (Pollak, 1893).

GENERAL CONSIDERATIONS IN REGARD TO TESTS.

This general review of the history of investigation and the sketch of our present knowledge of the structure and functions of the inner ear must be supplemented, if it is to be applied to the practical needs of clinical otology, by a study of the functional reactions in health and disease. It is evident that even the most minute and elaborate tests of patients presenting symptoms of labyrinthine involvement can result in accurate knowledge of the underlying morbid conditions and lead to a definite diagnosis only if we have a fairly constant and definite basis of normal reactions from which to measure given variations. It may be said here, by way of parenthesis, that there still remains a great deal to be done in this field. A large number of carefully conducted tests on normal individuals is needed to show the reactions to rotation, caloric, galvanic, and postural irritation, the physiologic limits of balance, the determining factors of vertigo, and so on. Even then the subjective quality of many of the reactions, for instance vertigo, dizziness, loss of equilibrium, will tend to vitiate accurate conclusions by the interjection of the personal equation of both patient and observer. The elaboration of quantitative tests will of course be the ideal in this direction, and the reduction of all observations to a uniform basis will simplify the entire subject. As to the application of these tests in disease, there are of course, other points, pro and con, to be considered. Many of the tests,

such as those of rotation vertigo and of balance angle, require special apparatus, a trained observer, much time, and more patience, besides the good will and co-operation of the subject. It is more than doubtful whether the tiresome and occasionally disagreeable rotation tests could be applied in private practice or even to any great number of clinic patients. The possibility of aggravating symptoms already present or of fancied injury on the part of the patient must be borne in mind. Galvanic irritation should be applied with the smallest perceptible current strengths, caloric nystagmus should be elicited without any mechanical violence, and the possibility of sudden falling should be thought of at all times, not only for the reasons just mentioned, but because, as we shall see, all these methods produce reactions in normal individuals and the results of our tests would be anything but instructive if they were conducted so hastily and roughly as to produce results which could be due to wide-spread irritation or the coincident reaction of distant sensory or central organs. This will be considered more in detail in the description of the clinical examination and tests of the labyrinthine functions.

THE REACTION OF THE NORMAL LABYRINTH TO FUNCTIONAL STIMULI AND EXPERIMENTAL IRRITATION.

Barany's tests were on normal subjects, patients with cranial injury, and on twenty-four cases of labyrinthine unilateral destruction. Up to the 50th year, age has no influence on the duration of consecutive nystagmus after prolonged rotation (10 turns). In normal subjects the average duration is 40 seconds, but there are marked individual variations. In dancers who do not "reverse," and are accustomed to dancing only to the right, the nystagmus is decidedly less marked after rotation to the right. Those who dance equally well to the right and left react like normal subjects. In health as well as disease after-nystagmus is more marked and lasting after rotation to the left. Repeated rotation tests show no signs of exhaustion in the reaction to turning; active fixation while turning tends to inhibit nystagmus. It is, accordingly, more marked if dark glasses are worn during the test, and lasts two or three times as long. Increased rapidity of turning lengthens the period of nystagmus and causes larger and more frequent

excursions. The number of rotations up to 10 increases the duration of after-nystagmus, but with continued rotation (Barany has gone as high as 60), the latter again becomes less. After 20 or more rotations the after-nystagmus is followed by reversal (tertiary nystagmus) in the original direction of rotation, which is of central origin.* In many normal persons one or two turns are sufficient to cause after-nystagmus, if fixation-correction has been excluded by dark glasses. In case of destruction of the labyrinth of one side the nystagmus is always more marked and lasting toward the sound side. Smokers and neurasthenic subjects show increased irritability to rotation stimuli.

Ocular torsion or wheel motion of the eyes, first noted by John Hunter in 1786, which compensates for head tilting and persists until the vertical position is again assumed, must also be considered as a labyrinthine reflex for all but small angles of inclination, as noted previously. After destruction of both labyrinths in animals, this torsion does not take place, while it is markedly diminished, according to Barany, in deaf mutes and bilateral destruction of the labyrinth in normal beings. In unilateral destruction there is no alteration, but in unilateral irritation there is decided increase of compensatory ocular torsion. In normal subjects a tilting of the head of 60° corresponds to ocular torsion of 4° to 16°.

TESTS OF EQUILIBRIUM.—V. STEIN, 1895.

v. Stein distinguishes between static and dynamic tests, in accordance with the theory of Breuer and Mach. For the former he uses a movable inclined plane on which the patient stands. The angle of inclination is then increased until balance can no longer be preserved. Tests with this goniometer show that in the upright position normal subjects can be titlted backward 36 to 39°, forwards 26 to 30°, and tipped sideways 36 to 38° before they fall. When the eyes are closed the angle of fall is 3 to 4° less.

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*This "tertiary" nystagmus appears in the model as a gradual advance of the pointer to the vertical, from reverse position (secondary nystagmus) as the fluid comes to complete rest after rotation. It can be explained quite as simply as primary nystagmus by the local conditions in the semicircular canals, without recourse to the central organs for a basis of explanation.

Dynamic tests note the variations from a straight line or the tendency to fall in a certain direction when moving in various ways. The difficulty of preservation of balance and direction increases as we proceed from standing to walking and hopping, from the flat foot position to tip toe, from a support on both legs to a single point d'appui. The difficulties are still further increased by closing the eyes and by moving backward. By these progressive tests v. Stein and others believe it possible to detect slight disturbances of equilibrium which would otherwise escape observation, especially when the patients themselves are not conscious of them.

Normal subjects can stand with closed or open eyes, on both feet, for many minutes without marked swaying. Walking backward and forward, under like conditions, generally follows a straight line. In labyrinthine affections Romberg's phenomenon of swaying, and characteristic deviations from the straight line are frequently noted. The more difficult tests are not equally well passed by normal individuals. Practice and special skill account for the various reactions, and as Boenninghaus has observed, we may be in doubt in a particular case what we are to ascribe to individuality and what to disease. Yet we may assume that in simple clumsiness or lack of practice, the defect of static and dynamic balance will be the same on both sides, while in labyrinthine affections there is usually marked inequality. The following phenomena have been found characteristic, but not invariably present, in labyrinthine disease:

1. The affected side is the weaker. In standing or sitting the body inclines or falls to the diseased side. In walking or hopping there is a deviation to the diseased side, the patient does not stand securely on the leg of the diseased side, and the body can be more easily pushed over to the diseased side by the observer (Adler, 1894; Hinsberg, 1901; Krotoschiner, 1906).

2. The body muscles tire rapidly, so that in hopping, the jumps become shorter and shorter, until finally they "mark time", and are made on one spot (labyrinth jumps of v. Stein). (Fig. 9.)

3. Falling can be prevented by merely giving the patient a finger (Mann, 1907), as the slightest tactile sensations will compensate for the labyrinthine defect.

4. Falling is sudden, unexpected, and complete. The patient falls "like a log", without regard for possible injury and without the power of prevention or compensation, so that the necessary precautions should always be taken by those who institute these tests.

Finally it may be noted that tests with the dynamometer, the ergograph, and other instruments of precision show a decrease of muscular energy, tone, and muscle sense, and characteristic curves of muscular action, in labyrinthine disease corresponding closely to the results of animal experimentation.

NON-ACOUSTIC SYMPTOMS OF LABYRINTHINE DISEASE.

According to Barany, the diseases of the semicircular canals may be divided into three groups:

1. *Circumscribed labyrinthitis.* O. I. circumscript.—Symptoms due to irritation by hyperemia or toxic edema. Spontaneous nystagmus at intervals, elicited by head motion toward diseased side. Caloric test positive. Many diseases show this reaction. The most important are circumscribed labyrinthine suppuration, and fistula. Fistula symptom (nystagmus on air compression) may be elicited. Subjective sensations usually present. Marked vertigo, nausea, and occasionally vomiting. Partial deafness. *Rotation* toward diseased side increases spontaneous nystagmus, or brings it on after one or two turns. After-nystagmus to sound side slight. *Vertigo* present. *Disturbance of equilibrium.* Head turns away from diseased side, and with head upright, patient falls away from diseased side. (Attempted compensation.) If head is turned toward diseased side, patient falls backward, and vice versa. *Tonus* increased on diseased side. *Rotation* toward diseased side causes more marked and lasting somatic reaction, which may occur after one or two turns. *Recovery* with normally irritable static apparatus.

II. *Diffuse labyrinthitis.* O. I. P. A.—Loss of function due to *acute* and widespread suppuration, and indicated by the loss of caloric reaction. Total deafness. Spontaneous nystagmus to sound side, due to complete loss of function on diseased side and comparative excitation of remaining normal labyrinth is continuous, present in all positions, and tends to disappear. Tinnitus and spontaneous nystagmus occasionally persist for some time. Rotation toward diseased side causes

slight somatic reaction, if any. Vertigo may be absent. Facial paralysis frequent. *Disturbance of equilibrium.* Tendency to fall or walk toward diseased side. Gradually corrected and compensated for by tactile and visual sense. *Tonus* markedly increased on diseased side. *Recovery* with unilateral loss of static function and loss of reaction to rotation.

III. *Chronic diffuse labyrinthitis.* O. I. P. C.—Loss of function due to old suppuration. Spontaneous nystagmus to sound side no longer present. Cerebral compensation and adjustment of temporarily disordered balance of labyrinthine stimuli. Caloric nystagmus absent. This may occur in cases which have progressed slowly from the start, with a subchronic course, nystagmus and vertigo never having been noted, and in cases of long standing labyrinthine suppuration with widespread destruction. Fistula symptom absent. *Rotation.* In II. and III. there is marked after-nystagmus to sound side. *Vertigo* absent. *Disturbance of equilbrium* shows gradual compensation. *Tonus* gradually restored, but muscular weakness remains.

SENSE OF BALANCE.

The sense of equilibrium and power of balance in human beings is a variable factor. Probably not congenital, as shown by the unstable equilibrium of small children, even when muscular power is great, and by the slight tendency to vertigo, which is rarely noted before the eighth year. Children are not subject to sea-sickness, can swing without becoming dizzy, or whirl in dancing or at play much more freely than adults. There are probably differences in the two sexes and at different ages. Neurasthenic or hysterical individuals are particularly susceptible to vertigo and nausaa. Occupation and training are of importance, and in tests of patients with labyrinthine disturbance it is well to ascertain what movements could formerly be performed correctly. There is a tendency for normal individuals to circle to the right in walking, and turning to the right especially in dancing is comparatively easy for the right-handed. Reversing is difficult for many, and this faculty should be inquired into. The tactile sense, muscle sense, tonus and sensations in the viscera combine to form one large uniform source of sensations as to our position in space. Visual sensations, sensu strictiori, and sensa-

tions of ocular muscle innervation, form a second. The third, labyrinthine sensations or disturbances, are generally subconscious and for that reason externalized and interpreted by the sensorium as sensations of objective or subjective motion. Normally, all changes of position or of equilibrium are associated with a sense of change in the field of vision, of the position of external objects as to us, an external disequilibration as opposed to the internal. Vertigo is produced when there is contrary innervation or discrepancy between the two sensation states. Thus even rapid and irregular motion of objects in the visual field will of themselves not cause vertigo, whereas very slight displacements will suffice when there is an absence of the usual corresponding sensations in the ocular muscles or those of the body.

OBJECTIONS TO BREUER-MACH THEORY AND V. STEIN'S TESTS.

Objections to conclusion from animal experiment, particularly section of the semicircular canals, as well as to deductions from the phenomena of their disease, in so far as it was assumed that these symptoms were evidence of local changes, were made from the beginning of investigations in this field. It was repeatedly claimed that the results were vitiated and the facts obscured by complicating disease or accidental injury of contiguous cerebral structures. Thus Hitzig (1871) believed that the phenomena of galvanic irritation nystagmus were due solely to cerebral irritation. Disproved by cocainization of the ampulla, and by observations on deaf-mutes. Boettcher (1875) laid stress on possible injury by traction on the nerve trunks in semicircular canal experiments. Weber-Liel (1880) considered brain irritation through changes of pressure in the aquaeductus cochleae sufficient explanation. v. Bergmann, in the same year, attributed Meniere's complex in the case of a fissure through the labyrinth to coincident traumatism of the brain. Lucae and Baginsky, judging from the effects of the air-douche and of injection of fluids into the middle ear, claimed that transmission of external pressure to the cerebrospinal fluid and consecutive central irritation or injury were the principal factor in disturbance of equilibrium. Baginsky in fact claimed that Meniere's complex was invariably dependent on cerebral lesions. Steiner (1888) claimed that all observed disturbances of motility could be explained by traction on the trunk of the auditory nerve, and that the phenomena were forced movements, not balance-

compensations. Other investigators claimed that the varied data of animal experimentation were due to faulty observation, and that with careful section of the semicircular canals there are no disturbances of equilibrium whatever. (Boettcher, Lussana, 1872.) There may be extensive disease of the labyrinth without a trace of disturbance of equilibrium (Lucae, 1882). Kiesselbach and Steiner experimenting on fishes and lizards, were unable to find any disturbances after injuring the semicircular canals. J. D. Richards (Treatment of Purulent Affection of the Labyrinth, ANNALS OF OTOLOGY, etc., Sept., 1907) claims that sound waves affect the non-acoustic as well as the acoustic labyrinth, and gives, as a practical example, a case in which after exenteration of the entire labyrinth, loud musical sounds produced intense dizziness. This merely proves again what was well known, that the central trunk of the eighth nerve transmits acoustic as well as equilibrational stimuli, giving rise to cerebellar sensory reactions, and does not in any way invalidate the theory of the non-acoustic function of the labyrinth. Other objections raised by Richards on the basis of a case in which disturbed equilibrium, vertigo, vomiting and nystagmus were relieved by removal of semicircular canals, vestibule, and lower half of the first cochlear whorl, are due to his having confounded evidence of irritation with loss of function. The removal of a source of irritation and of false localization would naturally be followed by disappearance of double or faulty sensation. Richards recurs to the old theory of concomitant injury of central organs or of centripetal nerves which he resurrects under the name of "peripheral irritation," to which the interior of the labyrinth is supposed to be subjected in removal of parts of the labyrinth. His deduction that nystagmus and other reactions are not entirely due to the loss of parts of the labyrinth, because under anesthesia mechanical irritation of the interior of the vestibule or on the ampullary areas may produce nystagmus, is faulty, inasmuch as anesthesia does not in any way exclude the function of the organ of equilibration. Against the practical application of v. Stein's tests, Richards states they are subject to numerous errors, as, irregularity in length of limb, a badly arched foot, fear of falling, and claims that a patient if he be right-sided (handed) attempting to jump in a straight line, will deviate to the left and eventually tend so

to fall if the eyes are closed. In the hopping test, the patient is not in contact with the floor long enough to gain a correct subjective appreciation through muscle sense of his proper relation to the objects about him. The disconcerting of muscular sense and suppression of orientation inherent in v. Stein's tests, create inequilibrium, so that if disturbed balance appears during the test we are at a loss to know what factor is responsible; in other words, it is not a differentiating test. In answer to these objections it can merely be said that they are not borne out by the results of practical experience. Normal individuals, whether flat footed, right or left handed, or otherwise asymmetrical, do preserve normal balance and walk straight forward or backward without difficulty, whereas, as shown by numerous observers, and again recently by Eagleton, labyrinthine disease does produce characteristic disturbances. No disequilibration is produced by v. Stein's test except the pathognomonic disturbance which we aim to elicit, and as to the tactile sensation during hops, if the length of time between them were insufficinet for orientation, we should all lose our balance when hopping, and there would be no tendency to fall to one side in preference to another. Richards' objections to theory and practice of equilibration and nystagmus tests are not well founded.

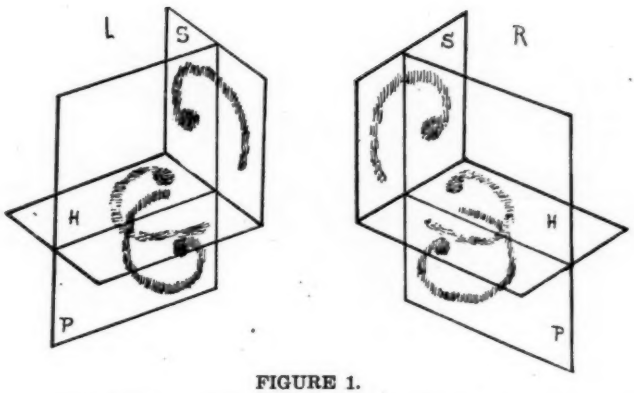

FIGURE 1.

Diagram showing relations of the semicircular canals (after Ewald). S, S, plane of superior or anterior; H, H, of external, horizontal; P, P, of posterior semicircular canals, seen from behind.

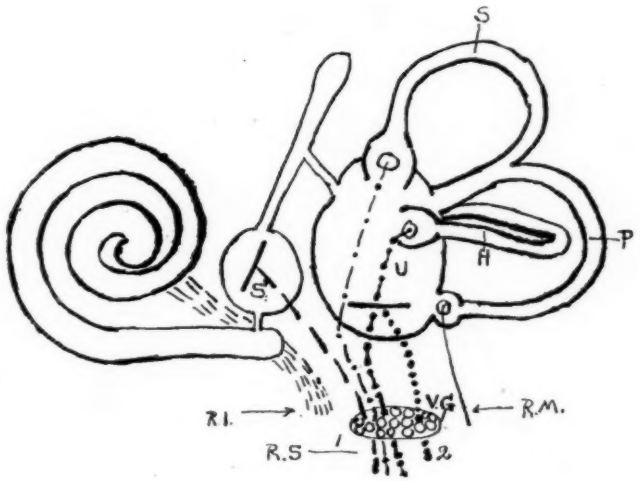

FIGURE 2.

Membranous labyrinth (left) from outer side. S, superior; P, posterior; H, horizontal semicircular canal; S^1, sacculus; U, utriculus with vertical and horizontal macula, respectively; R, I, ramus inferior (cochlear branch) of VIIIth nerve; R, M, ramus medius, from posterior ampulla; R, S, ramus superior; 1, first branch from macula sacculi, superior, and external ampulla; 2. second branch, utricular, from macula utriculi; V, G, vestibular ganglion.

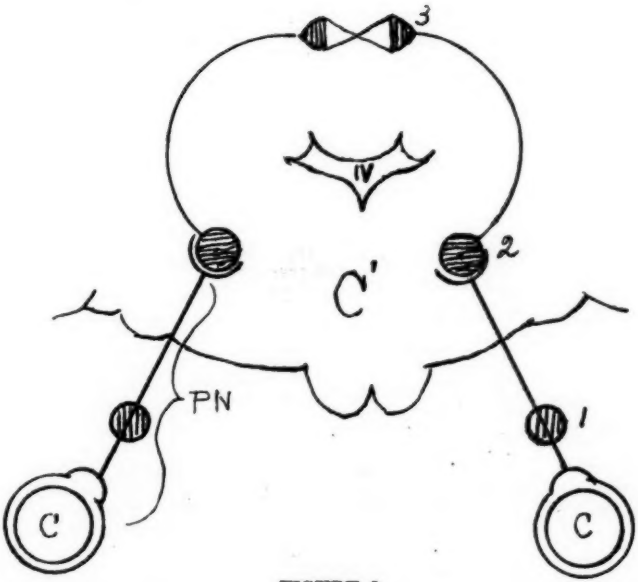

FIGURE 3.

Central course of vestibular nerve (after Boenninghaus). Diagrammatic cross section through pons and cerebellum. C, C, semicircular canal (vestibular apparatus); 1, vestibular ganglion; 2, dorsal nucleus of VIIIth nerve; 3, nucleus tegmenti; P, N, peripheral neuron; C^1, cerebellum; IV, fourth ventricle.

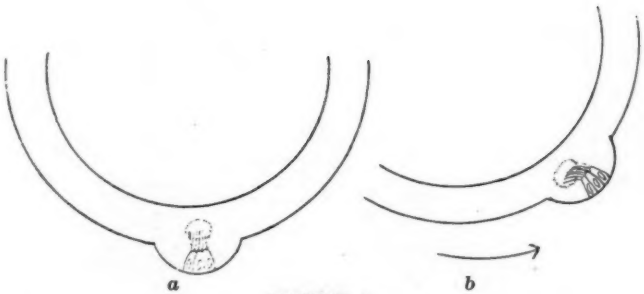

FIGURE 4.

Diagram of the action of inertia on sensory terminals in ampullae. a, canal and ampulla at rest; b, in rotation. In the latter the hairs of the sensory epithelium are dragged backward and irritated by the lagging of the endolymph.

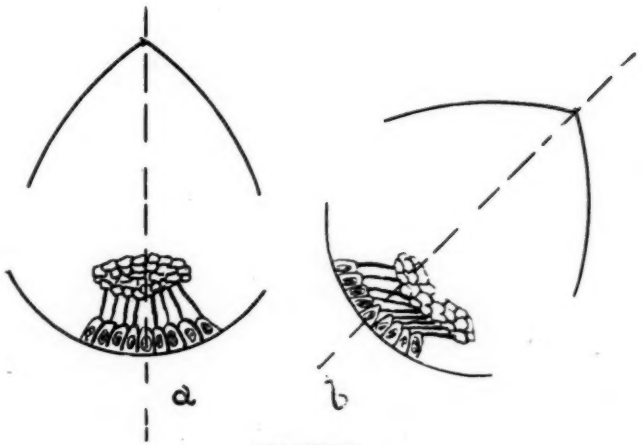

FIGURE 5.

Diagram of the action of gravitation on the sensory terminals of the vestibular organs (sacculus and utriculus). a, Vestibular organs at rest; b, inclined. In latter position the otolith plate causes by its weight and dragging, a distortion and irritation of the sensory hairs on the macula.

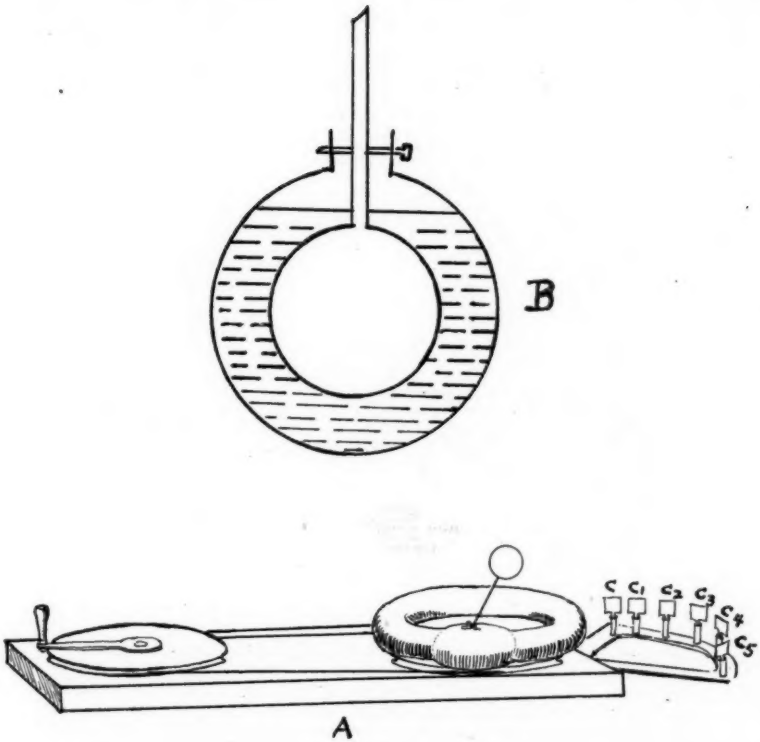

FIGURE 6.

Model showing mechanical principle of function of semicircular canals. A, Turn table. At right, a hollow ring of ¾ inch rubber tubing, nearly filled with glycerin and water, in which a small pendulum is suspended. B, Cross section through tube. Detail of pendulum. Beyond the table there projects a tin quadrant to demonstrate nystagmus. C-C^5, pegs covered with rubber tubing in which cardboard discs have been stuck to indicate external objects passing in rotation. On turning the disc, the fluid in the tube lags behind, at first, and holds back the pendulum, causing the indicator to tilt forward sharply in the direction of rotation. As the motion becomes more uniform, the indicator tends to stand upright. On cessation of motion the indicator reverses sharply, showing the momentum of the fluid in the tube, and the physiologic basis of sensation of reversed rotation. Acceleration or retardation of the disc is shown at once by an inclination of the indicator in the direction of turning.

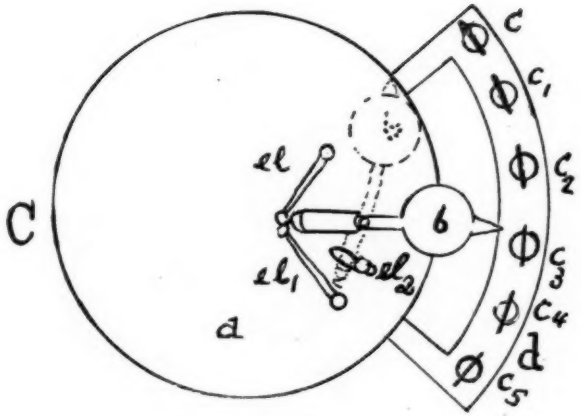

FIGURE 7.

Model showing mechanism of rotational nystagmus. C, Rotating disc; d, quadrant attached to base; C-C^5, pegs representing external objects, in which pointer, b, representing visual axis, engages; el, el^1, elastic bands to bring visual axis back to normal; el^2, attachment for forced abduction, as shown at b with dotted line. On revolving disc, representing motion of external objects while subject is turning, the pointer is held and caught a moment by each object in turn, springing back with a rapid recovery movement to normal position. On forced abduction with line of sight at a sharp angle with line of objects, the pointer is held much longer, the nystagmus, accordingly, appears more marked, when looking toward source of motion, and tends to disappear when pointer is in opposite position.

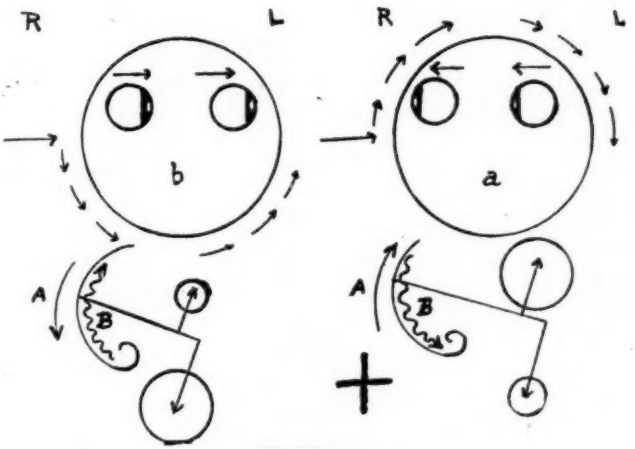

FIGURE 8.

Diagram showing direction of nystagmus, head-turning, and subjective sensation of motion, caused by caloric, mechanical, or galvanic stimulation. a, b, Face seen from in front, indicating direction in which eyes are turned, nystagmus; a, positive stimulation,+as by syringing right ear with hot water, turning patient to right, compressing air in right auditory meatus, or closing galvanic current with cathode on right ear. Nystagmus to R (small circle with enclosed arrow), head turning to opposite side; L (large circle with enclosed arrow). Movement of endolymph (serpentine arrow at B) strong, toward ampulla, away from source of irritation and from direction of real or sensory rotation, toward opposite side, giving subjective sensation (long curved arrow at A) of rotation to R, irritated side. b, Negative,—stimulation, as by cold water syringing of right ear, rotation to left, rarefaction of air in right meatus, anode closure on right ear. Signs as before.

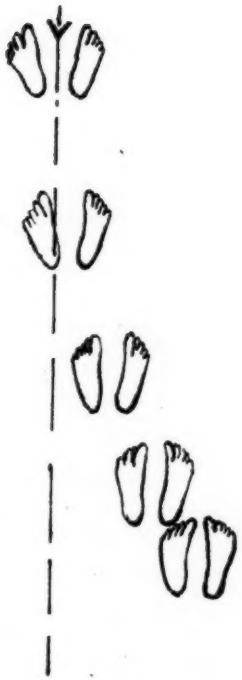

FIGURE 9.

"Labyrinth Hops," v. Stein. Hopping backward, in direction of arrow, in disease of right labyrinth.

XXVIII.

CHRONIC RECURRENT SUPPURATIVE OTITIS MEDIA AND ITS RELATION TO MASTOID AND INTRACRANIAL COMPLICATIONS.*

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PHILADELPHIA, PA.

INTERMITTENT OTIC DISCHARGE MORE DANGEROUS THAN THE CONSTANT VARIETY.

In considering the subject of chronic recurrent suppurative otitis media, I will endeavor to demonstrate that this particular form of aural disease is relatively productive of more complications, intracranial and otherwise, than the continuous variety. I have been particularly impressed with the importance of this subject from the fact that the belief still persists, both among the profession and the laity, that an aural discharge which is intermittent is of little importance, the assumption being, therefore, that the underlying disease is never dangerous to life. This fact clearly illustrates that the pathology and prognosis of these conditions are not generally understood, notwithstanding the marked progress which otology has made in this direction during the past decade.

OUR DUTY TO THE PROFESSION AND THE LAITY.

For years past many of us have been persistent and untiring in our efforts to impress upon the profession the importance of aural disease, and to indicate the obligation due our patients in the prompt recognition and treatment of the same. Like all weighty medical problems, the evolution has been slow, but we have at least established a fairly good general conception of the acute diseases, their dangerous complications and oftimes tragic sequelae. As the general surgeon has persistently urged, year after year, the necessity of early operative interference in appendicitis, so must we labor to secure a proper realization of the actual dangers of a discharging ear.

*Read before the American Laryngological, Rhinological and Otological Society, at Pittsburg, Pa., May 29, 1908.

Furthermore, we must not only recognize this condition, but must properly appreciate its gravity and demand the adoption of prompt and efficient measures for its relief.

This widespread indifference to a very grave problem is the natural outcome of continued apathy on the part of the medical profession, and if the Fellows of this Society are to conserve her honorable traditions, it becomes the immediate and imperative duty of each and every one of us to spare no effort, however self-sacrificing, to place before the profession some plain facts relative to the ever-present dangers of an otorrhea. We must, therefore, accept the obligations and responsibility created by such circumstances, and resolve to diffuse, in the most effectual manner at our command, such information as will throw proper and responsive light on this subject before the great body of practicing physicians, who will in turn be moved to action and will thereby become the legitimate instructors of their patients, the laity.

VALUE OF PROPHYLAXIS.

In this connection I wish again to take the opportunity of pointing out the great value of prophylaxis as applied to mastoid and intracranial involvement, complicating acute tympanic disease. In the primary stage we find a simple hyperemia and swelling of the mucosa, together with some seromucous exudate. At this particular period probably four-fifths of all cases will yield promptly if the well-known simple measures for their relief are immediately applied, whereas the vast majority of all serious complications are directly traceable to the unpleasant fact that these primary measures have been partially or wholly neglected.

I am not unmindful of certain cases complicating the exanthemata, influenza, etc., which seem to be virulent and purulent from their very inception, but even in such cases a free incision of the membrana tympani before bulging occurs will usually prevent serious involvement of the accessory cavities and adjacent structures. Such clearly defined facts should, therefore, convince us that a discharging ear, whether of the acute or chronic form, is frequently the result of woeful ignorance or indifference, and that the subsequent mastoid and intracranial implications are mainly chargeable to the same cause.

At times we are confronted with the statement that an

otorrhea has existed for many years without producing untoward symptoms. So long, therefore, as the patient experiences no actual suffering he is content, often by the advice of his physician, "to let well enough alone." This viewpoint is quite natural and excusable on the part of the patient, but it does seem inconceivable, in this day of progressive and preventive medicine, that those charged with and professing to conserve the public health, should so far forget their obligations or misinterpret their humane duty.

As a body, those practicing the learned profession of medicine are among the most honorable, and certainly the most self-sacrificing men to be found in any calling; consequently, this lackadaisical attitude toward aural disease that we too often meet with can only be attributed to misconception or misinformation as to the worth and importance of the subject. Such being the case, I again wish to proclaim this society, individually and collectively, the disseminators of such information or instruction as may be necessary for the betterment of our profession, which, when viewed in the light of civic duty, must be accepted as an imperative obligation due to our fellowman.

ILLUSTRATIVE CASES.

In order to properly illustrate the destruction that frequently accompanies a recurrent suppurative otitis media, I will briefly relate the histories of three typical cases, which I have recently operated upon.

CASE 1. *History.*—Male, T. W. D., aged 44 years. There is some doubt as to whether he suffered from aural disease early in life. During the past three years he has had a recurrent suppurative otitis media of the left ear, the primary cause of which is unknown.

The patient came under my care at the Jefferson Medical College Hospital, April 3, 1908, stating that for the past three or four days he had suffered very severe pain, not only in the ear and over the mastoid process, but also on that entire side of the head, so much so that hypodermic injections of morphine were necessary to relieve his suffering. The history furthermore states that these acute exacerbations occurred about every three or four months. In the interim the ear would be entirely dry and free from pain. Prior to the last attack, however, he was free from all aural symptoms for about eight consecutive months.

The examination showed the external auditory canal to be filled with a copious, brownish-yellow, foul-smelling pus. There was a good-sized carious opening through the posterior wall of the external auditory canal, and communicating with the mastoid antrum, through which pus was escaping. There was much swelling, redness and tenderness over the mastoid process, extending upward to the superior border of the temporal bone, anteriorly to the outer canthus of the eye and posteriorly to the occipital protuberance. Pressure on this swelling would force a large quantity of pus through the carious opening already mentioned into the external auditory canal. The posterior half of the membrana tympani was destroyed; the anterior portion, however, remained intact and was quite normal in appearance.

Patient's temperature at this time registered 103°. Operation was advised and refused. The following day he returned, still suffering, the temperature rising to 104° at noon. Operative interference was strongly urged, but again refused. The next day he came again, at noon, his temperature at this time registering 105 1-5°. He was not able to secure sleep or relief from pain even under the influence of hypodermic medication. He now readily consented to an operation, and was admitted to the hospital for this purpose.

Operation.—A free incision through the soft parts evacuated a large quantity of pus. This incision was carried from the tip of the mastoid to about one inch above the superior border of the auricle, and then forward to the outer canthus of the eye for a distance of about two inches. A transverse incision on a level with the mastoid antrum was carried almost to the occipital protuberance. This large field of operation was necessary on account of the extensive osteomyelitis present. In this particular case the extension from the diploe, which is usually first involved in cases of osteomyelitis, was external rather than internal, probably taking the course of least resistance. The entire external plate of bone was very dark, in some spots almost black, with numerous points of hemic ooze. All this area of necrotic bone was removed. There was a large carious opening in the mastoid process a little below the level of the antrum. The remaining part of the process was also discolored and showed many bleeding points. The necrosis was so great that the whole process was removed, including the tip. The sinus, as so frequently occurs

in these chronic cases, was situated rather superficially and far forward, so much so that part of the posterior auditory canal acted as the anterior bony wall of the sinus. The necrotic process had exposed the entire sinus, which was bathed in pus, and covered with a thick layer of what seemed to be healthy granulation tissue, with the exception of one part. The granulation tissue was removed on account of some bleeding, and this bleeding was increased by the removal of this tissue, although no opening in the sinus could be found. The dura was largely exposed, both through the tympanic roof and the antrum. The discoloration of the dura through the antral opening was so marked that it suggested the possibility of some intradural abscess formation. A hypodermic needle was introduced for the purpose of diagnosis, but with negative results.

The septic temperature ranged from below normal to 106°, and continued for some days after the operation. The man at times became quite delirious, and the whole picture resembled one of septic sinus thrombosis. My own conviction, and the judgment of the various consultants, favored the opinion that the man was suffering from a general septicemia. Hence, no further operative interference was resorted to. The wisdom of this conservative decision was borne out by the fact that after the patient had passed through superficial multiple abscess formations in various parts of his body, he made a complete recovery.

Blood cultures and differential blood counts were made at various stages of this patient's illness. The former demonstrated the presence of the staphylococcus pyogenes albus, and the latter showed, as might be expected, a marked hyperleukocytosis and a decided increase of the polynuclear percentage. This relative ratio between the hyperleukocytosis and the polynuclear percentage continued throughout his illness, clearly demonstrating the value of the blood count in all septic conditions.

Frequent examinations of the eye-ground gave negative results.

One point of interest and importance to which I especially wish to direct your attention, is the fact that we can hardly conceive that all the bone destruction shown in this case could have occurred in the three or four days which marked the acute exacerbation of the chronic recurrent suppurative otitis

media, and yet the patient and his family claimed that for eight months prior to the date of this acute exacerbation he did not suffer the slightest inconvenience from his ear and that the same remained entirely dry during that period.

This brings up for consideration the extensive pathologic changes that apparently take place without causing the patient any discomfort. We are frequently surprised at the extensive necrosis met with in operating for the relief of mastoid disease. This remark applies both to the acute and the chronic form of mastoid disease, but of course is much more common in the latter. From this experience, especially during epidemics of influenza, I am convinced that bone destruction does sometimes occur with little or no inconvenience, until a certain point has been reached. In other words, this furnishes us an additional argument in favor of comparatively early operative interference.

The fact that this patient was able to attend the dispensary service on three consecutive days, with a temperature ranging from 103° to 105°, would indicate that he possessed an unusually good constitution, and that he could not have had any serious intracranial involvement.

CASE 2. *History.*—Another case which well illustrates the point under discussion is that of H. D., aged 18 years, who suffered from a recurrent suppurative otitis media of the right ear from early childhood, the trouble recurring at intervals ever since. At times the discharge would cease for a period of about one year, the membrana tympani completely regenerating in the interval, the pain, however, being much worse during the period of cessation of discharge. Whooping-cough is the only disease from which the patient ever suffered, and this is said not to have had any influence on the aural condition.

An acute exacerbation occurred about two years ago and was very severe, the discharge lasting until he applied for treatment at the Jefferson Medical College Hospital, October 11, 1907. Examination at that time revealed the presence of a slight discharge in the external auditory canal escaping from a small perforation in the anterio-posterior part of the membrana tympani, the remainder of the membrane being intact and looking fairly healthy. The Eustachian tube was patulous, an increased amount of secretion being forced through the perforation by the use of Valsalva's method of inflation. Deep pressure over the mastoid process elicited some tenderness.

In order to provide for better drainage, the membrana tympani was freely incised and the patient placed on the usual line of treatment.

November 23, 1907, or about five weeks later, the drumhead was found to have entirely regenerated and was quite normal in appearance, the patient, however, complaining of severe pain over the mastoid process and the right side of the head, although, with this exception, there was not the slightest evidence of the classical symptoms of mastoid disease, as evinced either by redness or swelling over the process or by any inflammatory condition of the drumhead. There was, however, some drooping and slight redness of the superior and posterior wall of the external auditory canal.

When the patient first applied for treatment, the mother was advised that an operation would probably be necessary. The condition had now improved to such an extent that we advised deferring operative interference. This did not meet with the approval of either the patient or his mother, the latter stating that the boy had been going through this process practically all his life, and she was convinced that the recurrence of pain on that side of his head would soon be followed by a reappearance of the discharge and that at such times he usually became quite ill, with high fever, severe headache, nausea, vomiting and great prostration.

Operation.—Heath's operation was performed on December 6, 1907. Here again the mastoid was found to be of the diploic variety, the accompanying osteomyelitis extending back almost to the occipital protuberance. This, of course, offered a ready explanation of the pain on pressure over the mastoid and posterior to the same. As in the former case, extensive necrosis was found, especially over the sinus, exposing the same for almost its entire extent.

The sutures were removed and the dressings changed six days after the operation. The wound was dressed every second day until January 2, 1908, when the patient was allowed to leave the hospital apparently well. On February 15, 1908, or about two months after the operation, the patient returned, the discharge having entirely ceased and the hearing having shown marked improvement. On April 17, or about four months after the operation, the patient again reported for examination. The membrana tympani was now entirely regenerated, and there was a complete cessation of the discharge.

The hearing had improved almost to normal, and he was practically free from all discomfort.

The result, in so far as the hearing is concerned, shows the importance of resorting to a Heath or a modified radical operation in all suitable cases.

The destructive nature of a recurrent suppurative otitis media, to which little or no attention is usually paid, is well illustrated by a patient from the far West on whom I recently operated.

CASE 3. *History.*—Married woman, aged 38 years. Without apparent cause she has had a recurrent suppurative otitis media of the left ear practically all her life. Up to eight years ago the discharge would appear about every two months. At that time, however, it ceased until nine months ago, when the acute exacerbation was ushered in with characteristic headache, nausea and great lassitude, extending over a period of several days, until the discharge again appeared, which in turn relieved the pain considerably, as so often occurs. The otorrhea, however, had continued during this entire nine months. In June, 1907, she had an acute suppurative otitis media of the right ear, which was the first time this ear had been involved.

The patient consulted me April 7, 1908. She was found to be suffering severely with headache, confined to the left side. There was no swelling over the mastoid process, but she suffered from rather severe pain on gentle pressure over the mastoid, and extending for some distance posteriorly. There was a copious discharge escaping from the external auditory canal, of a brownish-yellow character, streaked with blood and very offensive. The entire membrana tympani in this ear was practically destroyed, as well as the ossicles, which, however, were said to have been removed on a former occasion. The superior and posterior wall of the external auditory canal was very red, extremely sensitive, and drooping. The hearing was practically nil.

Operation.—In this patient we found the cortex exceedingly hard, it being flint-like in character. The sinus was pushed far forward and exposed by the necrotic involvement. There was some necrosis of the bony roof of the mastoid antrum, which, on being removed, evacuated considerable pus from an extradural abscess.

The radical operation was performed in this case. The

patient made an uninterrupted but rather slow recovery, on account of suffering from an intercurrent attack of purpura hemorrhagica with hemophilia, the bleeding taking place at many points over the entire surface of her body, but more especially in the throat and gums. Although no undue hemorrhage was met with at the time of the operation, there has been a tendency since that time to have some bleeding from the mastoid cavity. On the other hand, up to the present time there has been no recurrence of the purulent secretion. The vertigo from which she suffered prior to the operation has continued at intervals. This may be accounted for by some possible hemorrhagic invasion of the labyrinth.

EXACERBATIONS CAUSED BY INFLUENZA.

Of seventeen mastoid operations performed by me in six consecutive days, during the second week of January, 1908, seven were of the recurrent variety. All showed extensive bone destruction, exposing either the sinus or dura or both. Two led to the evacuation of pus from a temporo-sphenoidal abscess, through the mastoid antrum route, and one assumed the Bezold's variety of mastoid disease. Previous duration of the disease ranged from twenty-seven months to thirty-one years. The acute exacerbations occurred in two cases about every two months, and in the others about every two years or more. Four of the patients had not noticed any discharge from the ear, nor experienced the slightest discomfort from the same for a period of three years or more. In one patient the ear had not discharged for six years when a sinus thrombosis developed. In all these patients the acute exacerbations were complications of influenza.

Of about one hundred mastoid operations recently performed, twenty-one were of the recurrent variety. Of this number, eleven showed unexpected necrotic exposure of the sinus or interior of the skull. Two died during the acute exacerbation, one from an old encysted temporo-sphenoidal abscess, which ruptured as the result of an injury to the head, and the other from an infectious meningitis. In this latter case free pus was found in the cavernous and petrosal sinuses, the lateral sinus being apparently uninvolved. This patient enjoyed good health until his final and fatal attack, his previous exacerbation occurring more than twelve years before. The primary ear disease developed from epidemic measles forty-two years prior to this date.

During an attack of suppurative otitis media, especially when long-continued, the mucosa lining the tympanic cavity and accessory cavities first undergoes an ulcerative process, and this in turn becomes macerated and peels off, leaving a greater or less area of exposed osseous surface. With the protective and nutritive coat removed, the bone becomes an easy prey to the destructive influence of pathogenic microorganisms Consequently it is hardly to be wondered at that so much necrosis of the bony structure should occur in such a large number of suppurative cases.

The location of the perforation in the membrana tympani would seem to offer some points in the diagnosis of serious intratympanic as well as mastoid caries. Although the rule is not constant, it nevertheless does occur with sufficient frequency to warrant the assumption that perforations situated superiorly and posteriorly in Shrapnell's membrane, and more especially if they exist in connection with drooping of the posterior and superior wall of the external auditory canal, are definite diagnostic indications of extensive caries of the tympanic cavity as well as of the mastoid process.

During suppuration the mucous membrane of the tympanic cavity shows hypertrophy, with the formation of vegetation or papillary growths, which may be large or small, discrete or confluent, extending along the inner wall of the cavity to the orifice of the Eustachian tube. As suppuration continues the epithelial cells desquamate and form large, whitish-yellow masses, which are composed of inspissated secretion and desquamated epithelium. These may be retained for an indefinite time unless infected by the staphylococcus, when they are converted into a foul-smelling discharge, consisting of bacteria and granular debris. Occasionally they assume a pearly white appearance and remain in the middle ear cavity as homogeneous masses or cholesteatomata.

The inward growth of the epidermis of the external meatus towards the tympanic cavity without the formation of cholesteatomata takes place more often than is generally supposed. On the other hand, secondary cholesteatomata are very often caused by the intrusion of the epidermis of the external meatus into the middle ear. They also develop in the middle ear itself, as shown by the presence of non-nucleated squamous cells. These cholesteatomatous masses may attain a considerable size without the least sign of caries or necrosis. On the

other hand, it must be remembered that these epithelial accumulations or growths are probably more destructive, both to the soft and osseous structures, and cause more serious mastoid and intracranial complications than any form of infection.

In long-standing cases the horizontal walls of the semicircular canals may become eroded and the internal structures become the seat of inflammatory changes affecting the aqueductus vestibulae and the aqueductus cochleae. This in turn may cause a cerebellar abscess, resulting from suppuration within the labyrinth, the pus having followed the auditory nerve and burrowed its way through the internal auditory meatus.

More especially during epidemics of influenza we meet a large number of serious mastoid cases, the cause of which cannot be definitely explained. In other words, from the patient's history and in so far as can be seen, the middle ear was not involved. In cases of this kind, the bacteria and their toxins undoubtedly must have reached the mastoid by a hematogenous form of infection.

As is well known, recurrent cases may remain quiescent for some years, when the patient suddenly complains of severe pain in the head and ear of the same side. There is general hebetude, followed by chills, fever, vertigo and vomiting, symptoms which frequently usher in some intracranial involvement.

In cases of a mild nature, or where an intracranial complication is neither present nor in the course of development, the intensity of the above symptoms will be more or less modified, as the severity of the attack is governed entirely by the underlying involvement.

DIAGNOSTIC VALUE OF LEUKOCYTOSIS.

In considering the bacterial invasion of the temporal bone and the adjacent structures, we often speak of the presence and value of a leukocytosis. In this connection it is well to remember that a leukocytosis is a perfectly normal condition in the healthy state, and is, in fact, nature's method of providing scavengers for the destruction of the numerous microorganisms that are constantly invading the human economy. This leukocytosis, therefore, must necessarily vary in degree, depending entirely on the quantity and character of the toxins to be destroyed.

In the great majority of cases a normal leukocytosis is quite

sufficient to destroy the hostile microbic incursion, but when the pathogenic organisms are numerous and virulent, a battle royal ensues between the leukocytes, which resent the hostile entrance into the possessions or domains of another, and the invading microbic army. In order to successfully meet and destroy the enemy, nature greatly increases the normal percentage of leukocytes, in which state the term hyperleukocytosis or leukopenia, which naturally signifies an entirely different condition. The normal number of leukocytes varies from 6,000 to 10,000 to the cubic millimeter. Hyperleukocytosis indicates an increase to 15,000 or more, and hypoleukocytosis or leukopenia is a decrease from the normal amount.

An examination of the blood is often an important link in the chain of symptoms which goes to make up the diagnosis in a septic process, but this examination must consist of the differential leukocyte count as distinguished from merely determining the presence of a hyperleukocytosis on the one hand, or leukopenia on the other.

The condition in sepsis, in the majority of cases, shows a decided increase in the leukocytes, but this is by no means constant, for as great an authority as Grawitz states that "not rarely active inflammatory processes with suppuration may be present without hyperleukocytosis occurring."

Leukocytosis gauges the combative powers an individual possesses and can use against the infection by which he is attacked, but it does not show the strength of that infection. Thus, in trifling infections in which the activity of the leukocytes is insufficient, and also in fatal cases where the system is overwhelmed by the toxins, hyperleukocytosis does not develop and even leukopenia may be absent. Facts such as these render the occurrence of hyperleukocytosis in sepsis an inconstant phenomenon. An early development and rapid increase in the number of leukocytes is decidedly in favor of septic disease. We have, however, a good index to the severity of the septic process in the relative percentage of polynuclear leukocytes. Normally there is present a variation of from 59 to 68 per cent of these cells, with a mean percentage of 61. Therefore, if a patient presents a blood picture in which the relative polynuclear percentage is above 80, it indicates absorption of pus from some part of the body. Pus is seldom found with a relative percentage of less than 80 unless the patient is a child, in which case it may be as low as 73. However, pus should

be suspected and looked for in all cases in which the polynuclear percentage is above 70.

In addition to the hyperleukocytosis there is at times another element which may be determined from the examination of the blood, namely, the demonstration of the special microorganism, often in almost pure culture. It is never sufficient to content ourselves with a single culture of the blood, but this should be repeated, and in addition various culture media should be employed.

The relations of the ear to sepsis are extremely important. Ponfick examined the ears in one hundred children whose ages varied from one month to four years, and who had succumbed to various diseases. The autopsies revealed that 78 times there was bilateral, and 13 times unilateral, otitis media. In only nine cases was the middle ear normal.

From the foregoing it would seem that we have at our command a valuable asset, which, when properly worked out, may throw much invaluable light on certain forms of sepsis. For example, in cases of recurrent suppurative otitis media, during the stage of apparent inactivity, or in other words, between the periods that mark the acute exacerbations, it may be possible, by an examination of the blood, to demonstrate that a retrogressive metamorphosis is in actual progress. The value of such findings would at once become apparent if we were enabled thereby to appreciate such conditions, even in the absence of certain localizing symptoms.

XXIX.

SOME SURGICAL PRINCIPLES ESSENTIAL TO THE CURE OF FRONTAL SINUS EMPYEMAS.*

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A review of the recent literature of the surgical treatment of the pyogenic affections of the frontal sinus shows clearly that much difference of opinion exists among operators of experience concerning the proper plan of procedure. The varying degrees of severity of the disease that may be present in the sinus, and the several accessory conditions that may occur in a given case, often make the choice of plan debatable; but in those cases of suppuration of the sinus where the symptoms are quite similar in every essential, it would seem that too great a difference of opinion exists among operators of apparently equal prominence and experience. Such a difference of opinion is unfortunate for those operators of less experience, who as a result, too often feel a sense of doubt and uncertainty when contemplating the performance of frontal sinus surgery. The diversity of opinion emphasizes the fact that this class of surgery is often difficult, and that ideal surgical measures have not yet been established. To some extent, no doubt, the present views concerning frontal sinus surgery are the result of the rather rapid evolution of the rhinologist from a condition of practice in which his chief duty, only a few years ago, seems to have been to cleanse and medicate the mucous membranes of the upper air tract, to that which today may require the execution of the most skillful surgical measures, involving not only the nose, but also the more distant and intricate structures of the entire head. This evolution from the erstwhile rhinologist who was a sprayer of noses to the present rhinologist who must be a high type of surgeon has in some measure been responsible for two sets of operators, viz., those who believe that all, or nearly all, cases of accessory nasal sinus suppuration should

* Candidate's thesis, American Laryngological Association.

be treated by the intranasal route, and those who hold that while the simple sinus empyemas may usually be thus successfully managed, when much actual pathologic change has taken place in the structure within the sinus or its bony environs, external surgery should usually be chosen.

The discussion which continues between that class of surgeon who maintains, as does Ingals, that 95 per cent of all cases of suppurative frontal sinus disease can be cured by the intranasal route, and that class which believes that the greatest certainty of cure, and the least danger to the patient lies in the methods of operation by external surgery, has brought forth some good arguments in support of their respective beliefs. The intranasal surgeon points out in support of his position the alleged facts that ample drainage, and the free admission of air into the diseased sinus will cure all except the worst cases, and that as a result of the concealed operative measures required, no unsightly scar remains. The chief argument of those who prefer external surgery and the only one they consider absolutely necessary to make is, that thereby only can perfect drainage be secured, and the diseased structures be completely removed. The more or less scar tissue which results from external surgery of the sinus, while always an objectionable feature, is by this class of surgeons regarded as a sequence absolutely necessary to a successful issue, and therefore entirely justifiable.

This paper is not, however, greatly concerned in the dispute between the two classes of surgeons, for both admit that a certain number of all cases of suppurative sinus affection must be operated by the external route, and that a certain percentage is also curable by intranasal procedures; but it is rather with the surgical principles essential to the cure of sinus empyema which should govern the method which is chosen, that we shall be most interested.

Good results from the practice of any method must depend largely upon the proper application of certain well established surgical principles. Aside from an aseptic technic, which should be as perfect as possible in every case, the principles upon which all successful sinus work must depend are: 1, the complete removal of all pyogenic structures; 2, provision for ample drainage of the operated parts; 3, heal-

ing the diseased cavity either by a healthy, non-suppurative lining, or by the complete obliteration of the space by means of granulation tissue. The true value of either the external or intranasal method of treatment of suppurative frontal sinus disease can, therefore, be excellently judged by subjecting each plan to the standard of requirements of the above surgical principles. Of course in addition to these principles, the length of time the patient must suffer or be inconvenienced by the necessary treatment subsequent to each, and the actual danger to life resulting, although not surgical principles, are nevertheless often the result of the application of correct or faulty principles, and must, therefore, in justice be considered. Let us apply these principles first to the method of treatment by intranasal drainage.

The efforts of operators like Ingals, Halle, Good and others to cure frontal sinus suppuration by the intranasal route, and the ingenious devices which they have perfected for entering and draining the frontal sinusus, should receive the heartiest commendation. Undoubtedly a large percentage of cases in which structural changes have not been extensive, and in which the anatomic formation of the sinus is normal, can be and are cured by the simple provision thus made for good drainage. When, however, anomalies in the anatomic structure of the sinus are present, and when extensive pathologic tissues have been formed within, drainage alone is not often sufficient to bring about a cure; for in most instances it is impossible to remove this diseased pathologic content of the sinus, or to correct its anomalous structure through the comparatively small opening which it is possible by this method to provide for the drainage. Moreover, in case of the frontal sinus it has been considered a settled point that it is not possible or safe to enter the sinus with instruments intended for the enlargement of the infundibulum, in a considerable percentage of cases. Mosher (Candidate's thesis for American Laryngological Association), after a study of the applied anatomy of the frontal sinus in 100 wet specimens, and 50 cleansed skulls, states that it is only by chance that the naso-frontal duct can be catheterized. Turner (Accessory Sinuses of the Nose) says, the variation in the frontal sinuses which have been described, sufficiently indicate that the passage of the probe through the anterior nares toward the frontal sinus may meet with some opposition. Occasionally a narrow or

tortuous duct, or the small size of the ostium frontale, may prevent the probe reaching the sinus. Beaman Douglass states concerning this point (*Laryngoscope,* May, 1904): "One is often mistaken in believing that he has entered the frontal sinus, when, as a matter of fact, his instrument is within an enlarged anterior ethmoidal cell (bulla frontalis). All who have extensively examined the frontal sinus either in the wet or dry state of the skull must have observed that in a considerable percentage of instances an anterior ethmoid cell projects itself into the frontal sinus either external or internal to the sinus orifice of the duct, and that the mouth of the duct lies partially under such cell. In this class of case catheterization of the frontal sinus, when successfully performed, must always result in the entrance of the instrument through the broken down ethmoidal cell wall, through which it has been forced. Douglass has, furthermore, pointed out the fact, which has also been observed by others, that the internal wall of the frontal sinus frequently lies so low above the orifice of the duct that any cutting instrument, even if successfully introduced through the nasofrontal duct, would immediately penetrate the cranial cavity and would most probably set up an infective meningitis. On this point Mosher states that if there is a sufficient basal relationship between the floor of the frontal sinus and the nose, a burr can be pushed up through the duct into the sinus, the burr running on a probe as a pilot. Concerning this plan of entering the sinus, Mosher observes that, on the cadaver, beautiful specimens can be made in this way, but that he is always afraid that the practice of the method will make cadavers of some of his patients. The literature of the subject, therefore, clearly indicates that few operators would be willing to place so high an estimate on the percentage of successful cases of intranasal enlargement of the duct as does Ingals, who, as previously quoted, states that 95 per cent of all frontal sinuses may be safely and successfully entered and drained by means of intranasal instrumentation.

It should be stated, however, in justice to the method in Ingals, that his latest instruments and plan of procedure for entering the frontal sinus through the nasofrontal duct are undoubtedly less dangerous than formerly (Ingals' paper before the New York Academy of Medicine, Section on Laryngology, Dec., 1907), for this operator now passes a

guide through the duct into the sinus, next by means of the x-ray determines that the position of the guide is correct, and then, when certain that all is safe, drives the burr, which cuts only on its anterior face, along the guide until the whole course of the infundibulum is enlarged and the sinus is entered.

Unfortunately the most perfect drainage which it is possible to secure by operative enlargement of the nasofrontal duct, does not always thoroughly drain the entire frontal sinus, and this simple procedure of enlarging the nasofrontal duct will not, therefore, cure every case even though of an uncomplicated, acute variety of sinus suppuration. As evidence of this, several anatomists have especially pointed out the fact that the frontal sinus is often partially, and more rarely almost completely divided by osseous septa in such a way as to preclude the perfect drainage of every portion of the sinus, even though the nasofrontal duct be widely open. Thus, Hoeve, in an examination of 100 heads (*Laryngoscope*, July, 1907), states that frequently an internal set of frontal sinuses was normally present, due to the middle sagittal septa being complete. Hoeve further states that occasionally a superior accessory frontal sinus was present in thick skulls, and that frequently posterior and external accessory sinuses were found during these examinations. The writer has seen one posterior accessory frontal sinus, and two external accessory frontal sinuses in cases of secondary operation which he performed because of failure to cure by intranasal method, and he has frequently found the external accessory sinus present during external operations in primary cases that seemed urgently to demand the surgical interference.

It must be clear to every operator of experience that intranasal methods of operation upon the frontal sinus are in accord with but one of the several essential principles of successful surgery, viz., the provision for better drainage; and that even this principle is often violated because of the frequent pocketing of the sinus as a result of the presence of coronal or sagittal septa within. It would seem, therefore, that it may be justly stated that there are at least three classes of frontal sinus suppuration that can not be cured by intranasal methods. These are: 1. Those in which the inner table of the skull arches above the sinus orifice of the nasofrontal duct so closely that, to enlarge this orifice through the intra-

nasal route by means of cutting instruments, would almost certainly endanger the life of the patient because of injury to the dura mater at this point. 2. Those cases in which more or less complete sagittal or coronal bony septa within the sinus prevent good drainage even though a large nasofrontal opening be provided. 3. The cases in which extensive pathologic changes have taken place in the mucous lining or osseous walls of the sinus.

Having briefly shown the limitations of the intranasal method of operation on the frontal sinus, when viewed from the standpoint of surgical principles involved, let us next estimate the value of the external procedure when subjected to examination by the same standard.

By following the external method only can the surgeon hope in any satisfactory measure to carry out the surgical principles of removing in an absolutely thorough manner all the diseased tissue involved. This is the method of certainty in this respect, and not one of guess work. No thoroughly trained surgeon is today at all satisfied that he has performed commendable work in the removal of diseased structures unless he actually sees, and therefore, knows, that he has in nowise disobeyed this first essential of all good surgery. Concerning the dictum that the most perfect drainage should be provided, it will probably be admitted by most operators that the external method of operating on the frontal sinus is superior in every way, for when either the anterior or inferior wall of the sinus is removed, or when both have been resected, as in the Killian operation, every means of access possible is provided for the removal of all the diseased structures which will subsequently interfere with the outflow of exudates. The external method furnishes, also, conditions which are more favorable than any other to the healing of the cavity by means of healthy granulation tissue, because, as will be subsequently stated, if this method of operation provides the best means of thoroughly eradicating the diseased structures, and establishes a thorough drainage for the operated parts, it necessarily follows that the most ideal conditions are provided for a healthy growth of the necessary granulation tissue.

The application of well established surgical principles to the external or intranasal methods of operation for the cure of frontal sinus empyemas seems, therefore, to offer little

ground for good argument in favor of the latter plan of treatment, after the disease has long since passed the acute stage, or after it is known in any case that the given sinus consists of a series of compartments rather than of a single, smooth cavity, all parts of which drain, funnel-like, toward the infundibular orifice.

Which of the several methods of external operation is most ideal when viewed from the standpoint of the application of correct surgical principles? The writer has had most experience with the Killian operation, and with the open method. The procedure devised by Prof. Killian seems more in accord with good surgical principles than any other method in which the external wound is immediately closed, and therefore if a primary union of the external wound is intended it should be the method of choice. This operation is also desirable from the reason that a minimum of scar is produced. Although the Killian operation is ideal in many respects, and especially is this true when the anterior ethmoidal cells are diseased, the writer believes, judging by his own limited experience, that when radical surgery is indicated in the cure of frontal sinus empyema, that the desired end is often most certain of attainment by following the so-called "open method" of treatment—that which has been advocated and extensively practiced by Coakley—for by this method greater opportunity is offered the surgeon to provide and maintain throughout the whole course of treatment the most perfect drainage; to foster by the art acquired by his experience the natural growth of the necessary granulation tissue; and should perverse or freakish behavior of the granulation structures at any time become manifest, at once to correct and to restore the same to a satisfactory and normal state.

The probable deformity resulting from the open method, has, of course, been an insurmountable objection to its employment in the treatment of frontal sinus suppuration, but the writer believes that a careful and prolonged experience in dealing with the healing of osseous wounds such as the accessory sinuses, by means of obliterating them with healthy granulation tissue, will in great measure enable the surgeon to avoid ugly and greatly depressed cicatrices. Perhaps nowhere in surgery does experience count for more in securing esthetic results, than in the case of closure of an osseous wound by the open method. The surgical principle will pres-

ently be stated that over-pressure will stunt or kill granulation tissue. Hence if the gauze packing which is commonly used in the treatment of the sinus by the open method is inserted with too much pressure the filling-in process is lengthened to such an extent that the epithelial structures will probably cover the surface of the wound before the granulation tissue has risen to the skin level, in which event a depressed scar is the inevitable result. A similar outcome may likewise occur from an undue energy on the part of the surgeon in a too excessive or a too frequent repression of the granulation tissue by means of cauterization. A study of this class of wounds during the healing process, and especially a study of the habits and behavior of granulation tissue is, therefore, a very necessary part of the surgical technic, and should provide a satisfactory means of avoiding ugly scars, in many instances at least.

In a personal experience of more than 500 mastoid operations, and 11 frontal sinus cases treated by the open method, the entire after-treatment of which the writer carried out in nearly every instance, opportunity was offered for rather conclusive observation concerning the behavior of granulation tissue under almost all conditions that are likely to occur during the healing of open wounds. The observations thus made have been of great service to the writer in securing a more rapid closure of the frontal sinus, and in avoiding to a great extent the resulting depression and ugly scars. The conditions under which healthy osseous granulation best thrives was noted, as well as the states of the wound which were most favorable to overactivity and necrosis of the granulation structures. The growth of this tissue in osseous wounds as thus observed seemed often freakish, and its behavior such that a useful interpretation of the same was frequently impossible. Nevertheless, in the majority of instances its development was most natural and vigorous under the following conditions: (a) When the wound was sterile, or at least in a great measure approached a state of sterility. (b) When the granulations were moistened only with the exudates which were present, but were neither submerged in secretion on the one hand, nor too greatly dessicated on the other. (c) When there was at no time a too pronounced or too prolonged pressure from the gauze or other dressing which had been inserted into the cavity. (d) When the primary operation on

the sinus had been thorough in every respect, and the operated osseous wound had been left smooth and free from any pockets capable of retaining subsequent secretion.

With a thoroughly prepared osseous wound as above described, clinical observation teaches that the subsequent behavior of the granulation buds may be entirely controlled at the will of the surgeon, who may by means of gauze packing, or the application of caustics, repress the growth of new tissue, if he so desires, until the whole cavity has been covered with epithelial structures, and the wound has entirely healed, leaving a maximum of depression and scar tissue. On the other hand it is entirely possible, in most instances at least, to secure in a satisfactory way, a type of vigorous granulation structure which will rapidly obliterate the sinus by filling it in to a level with the skin structures, which latter are then, and not till then, allowed to bridge across the wound. Healing of the sinus by the open method of treatment will, therefore, leave a minimum of depression and scar tissue, provided the surgeon will exercise that control over the granulation structures which experience has abundantly shown may be exercised almost at the operator's will. The desired pliability of the new tissue is based almost entirely upon the surgical principles of efficient drainage, absence of undue pressure upon the granulation buds, and a properly prepared wound to begin with.

Clinical observation of the processes of the healing of bone wounds by granulation also teaches that if adequate drainage is provided, and the wound is secured against exposure to outside pathogenic bacteria, a sufficient degree of sterility will usually be secured to encourage rapid and successful filling-in of the cavity. The surgical principles involved in the provision of such ideal drainage for this class of wound will also insure against the accumulation of exudates and the consequent harmful flooding of the new tissue. In sinus surgery, therefore, successful restoration of the cavity, or its complete obliteration, needs scarcely be expected in any case in which the external wound is immediately closed, unless the operator is absolutely convinced that the drain canal he has provided is ample to insure against retention of fluids and the inevitable infection of the same, should they be permitted to stagnate. Even though such an ideal drainage is established at the time

of the operation, if immediate closure of the wound is practiced two things may speedily happen to cause failure to cure. Those are, 1st, the too rapid closure of the drain canal by granulation tissue, and, 2nd, the formation of pockets through faulty behavior on the part of the granulation and the consequent retention of pus in the sinus, even though an adequate drainage canal remains intact. This latter condition is one which, in the writer's opinion, is responsible for the failure in many cases which have not improved, and in which secondary operations have, therefore, been necessary. It has already been stated that the behavior of granulation tissue, even when all conditions are apparently normal, is sometimes freakish and unaccountable. For example, the writer has often observed during the post-operative treatment of open wounds in the several head sinuses, that occasionally when all had been progressing perfectly well, if the patient be not seen for 36 or 48 hours, granulations of unaccountable size would spring up in some part of the wound and grow toward similar granulations in another part with such amazing rapidity that adhesion between the opposing buds took place, a wall was constructed across the healing cavity, and, before the surgeon was scarcely aware of what had occurred, some portion of the wound was already badly drained, the granulations rank and perhaps necrotic, and unless the faulty conditions thus established were promptly corrected, failure to heal the wound was inevitable. Of course, should such an occurrence take place in an open cavity, every part of which can be inspected at any time, failure should not result, because the error is easily detected and corrected; but when the external wound has been immediately closed at the time of the operation, or when intranasal drainage only has been practiced, the surgeon can only surmise what is going wrong, and is, therefore, helpless to correct any freaks of healing by granulation, except by means involving a secondary operation.

XXX.

A CASE OF ACUTE SUPPURATION OF THE LABYRINTH FOLLOWING ACUTE OTITIS MEDIA—OPERATION; RECOVERY.*

By Edward Bradford Dench, M. D.,
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While cases of acute suppurative inflammation of the labyrinth are not so very uncommon, the symptomatology of the affection is far from clearly defined. This especially is true of those cases which follow an acute inflammation of the middle ear.

I take the liberty, therefore, of reporting somewhat in detail the following case, on account of the rather unusual manner in which the symptoms presented themselves.

The patient was a man sixty-five years of age who, about two weeks before I saw him, had an acute attack of grippe, with an acute suppuration of the right ear. Spontaneous perforation occurred early, followed by profuse discharge. The drainage from the ear was insufficient, however, and eleven days before I saw him, the right drum membrane was incised by a competent otologist. Free drainage was established, but the patient had suffered ever since the beginning of the attack, with severe pain in the head, together with pain in the right mastoid region. The patient also complained of inability to sleep. This sleeplessness, however, could not be attributed to the acute inflammation in the right ear, as he had been suffering from insomnia for some time prior to the acute otitis.

When I first saw the patient, I found him rather excitable, complaining of severe pain in the head, particularly upon the right side. There was some tenderness over the mastoid antrum, over the tip of the mastoid, and over the mastoid emissary vein. This tenderness was elicited only on firm pressure, and was not very severe. There was a profuse, purulent discharge from the ear. Examination revealed a perforation in the antero-superior quadrant of the drum membrane, just beneath the anterior fold. A probe introduced through this per-

* Read before the Annual Meeting of the American Otological Society, Atlantic City, June 24, 1908.

foration detected roughened bone. The aural discharge was not examined at this time, but it had been examined by the attending physician, who reported that the smears showed a diplococcus, and that no streptococci were present. A blood count had been made, showing a normal leucocytosis. No differential count had been made.

When I first examined this case, I was inclined to believe that operative interference would be necessary. The temperature of the patient was practically normal at this time, and it had been so for several days prior to my first visit. As I have said, when I first saw the patient, I believed that immediate operation would be necessary. I saw the case frequently, however, and the mastoid tenderness disappeared. The discharge from the meatus diminished somewhat in quantity, but still retained its thick, purulent character. The patient was observed, at intervals, for one month. During this time the mastoid tenderness entirely disappeared, as did also the headache and sleeplessness. The patient was able to be about and take regular out-of-door exercise. When, however, he attempted any mental exertion he became very nervous and easily fatigued. Owing to the fact that the purulent discharge from the ear still persisted, in spite of the fact that all mastoid tenderness had disappeared, I determined to open the mastoid. This operation was performed one month after I first saw the patient. At the time of operation the cortex was found to be perfectly normal, and fully an eighth of an inch in thickness. On opening the cortex the entire mastoid process was found to be broken down and filled with granulation tissue. The internal table was entirely destroyed, and the lateral sinus ran through a mass of granulation tissue. The wall of the sinus had become softened, and the sinus was opened through the gentle removal of these granulations by means of the curette. Hemorrhage was easily controlled by means of gauze packing. The complete mastoid operation was done. A small epidural abcess was found just at the knee of the sinus, containing about half a drachm of thick yellow pus. After the mastoid operation, the patient complained of severe pain in the head, which prevented him from sleeping, and required moderate doses of sedatives. I attributed this pain to the blocking off of the lateral sinus, but owing to the extensive exposure of dura, and the fact that the case had progressed about six weeks prior to the operation, I also feared the pos-

sibility of a collection of pus within the cerebellum. All unfavorable symptoms, however, gradually disappeared, and the patient was able to leave the hospital and return home. The wound did well from the first, and in about five weeks after the operation, had contracted down to a comparatively small sinus, through which a probe could be passed into the middle ear. The discharge from the ear ceased almost immediately after the operation, and the ear remained dry for about two weeks. At this time, the patient contracted a slight cold, when the discharge from this external auditory canal reappeared and persisted. There was free through-and-through drainage from the posterior wound to the canal, but, in spite of this, the discharge from the canal never completely disappeared. About five weeks after the mastoid operation the patient was suddenly seized with a severe attack of vertigo. In walking there was a tendency to fall toward the unaffected side. This vertigo was attended with nausea and vomiting. When the vertiginous symptoms first appeared, there was no nystagmus, but twelve hours later there was pronounced nystagmus, this being more marked when the patient looked toward the healthy side, although there was slight nystagmus on looking toward the affected side. This latter symptom, however, disappeared quite rapidly, and nystagmus could then only be elicited by looking toward the healthy side. The temperature was normal on the night of the first appearance of the vertiginous symptoms, and the next day it reached 101°. The patient complained of considerable headache.

It should be stated that immediately after the mastoid operation several blood counts were made, and at no time did the polymorphonuclear count rise above 68%, while on most occasions it was below 60%. Immediately following the acute vertiginous symptoms, the polymorphonuclear count rose to 74.5%, with a leucocytosis of 13,400. A smear from the wound showed a few bacilli, a few diplococci without capsules, and some large and small cocci. The discharge did not seem to exhibit any particular virulence. The patient was seen in consultation by a prominent neurologist, who was at first inclined to believe that there was an abcess in the cerebellum. The reflexes at this time were somewhat exaggerated, and there were decided symptoms of meningeal irritation.

Knowing that all these symptoms could have been occasioned

by an acute invasion of the labyrinth, I refrained from any operative interference. Forty-eight hours after the appearance of the labyrinthine symptoms, the temperature became normal. The polymorphonuclear percentage dropped to 56.4% and the leucocytosis fell to 9,400. An examination of the fundus oculi simply showed some slight tortuosity of the veins. I might say that this same condition of the ocular fundus existed, a few weeks after the simple mastoid operation had been performed. As the examination was in each instance, made by the same man, he reported that there had been practically no change in the fundus oculi. Owing to the fact that the temperature had fallen, and that the blood count showed no indication of absorption of pus, and also, as the patient's symptoms were gradually disappearing, I decided to defer operation.

The patient gradually, steadily improved for the next six weeks. At the end of this time the mastoid wound had entirely closed, with the exception of a small sinus at the upper angle of the wound which led directly into the middle ear. The discharge from the ear, however, continued, and the probe introduced through the perforation in the drum membrane, still encountered roughened bone. An examination of the discharge from the ear showed streptococcus capsulatus.

During the six weeks following the vertiginous attack, the symptoms of disturbed equilibrium steadily improved. The vertigo now was so slight as to cause the patient but little annoyance. The nystagmus entirely disappeared, as well as the pain in the head, and the patient was able to sleep well at night. On account of the persistence of the discharge, however, I determined about seven weeks after the attack of vertigo, to perform the radical operation. At the time of the operation, I found a small eroded area in the horizontal semicircular canal. The lumen of the canal was opened, and a small amount of turbid fluid escaped. The oval window was then examined and, in order to establish free drainage of the labyrinth, was enlarged. In enlarging the oval window, a scale of necrotic bone was removed from the promontory, uncovering the lower and a portion of the second turn of the cochlea. The interior of the cochlea presented a reddish appearance, as though some granulation tissue was present there, although no pus could be detected. Thorough drainage was established between the opening in the horizontal semicircular canal and

the opening in the vestibule below the aquaeductus Fallopii. The posterior wound was completely closed, with the exception of a small opening at the lower angle, where a gauze drain was inserted. The external auditory meatus was enlarged by cutting a tongue-shaped flap from the concha, thus securing complete drainage of the entire cavity through the meatus. A meatal skin graft was applied.

Since the radical operation the patient has progressed favorably. The dizziness has entirely disappeared, there has been no nystagmus, and at the present time the radical cavity has completely dermatized.

The examination of the ear after the appearance of the vertiginous symptoms was interesting. A moderate whisper was heard, two weeks after the vertiginous attack, at 7 feet from the ear. The tuning-fork placed on the forehead, was heard better on the affected side, but the absolute bone conduction was diminished on both sides. The upper-tone limit was lowered to 4 Galton on the affected side. One month after the attack, and before the radical operation, a moderate whisper was heard at 5½ feet in the affected ear. Bone conduction was about equal on both sides, but the tuning-fork placed upon the forehead, was referred to the affected side. The lower-tone limit in the affected ear was elevated to 512 double vibrations per second. The upper-tone limit was 4 Galton. The tone limits on the unaffected side were normal.

After the operation, moderate whisper was heard at a distance of 6½ feet, and the vibrating tuning-fork placed upon the forehead was referred to the affected ear, although absolute bone conduction on the affected side was slightly diminished. The lower-tone limit on the affected side, was 512 double vibrations and the upper-tone limit was 4.5 Galton.

This case to me was interesting from several points of view: In the first place, the appearance of labyrinthine symptome, in spite of free drainage of the middle ear through a posterior opening. We usually expect to find labyrinthine symptoms developed only in cases where there is some obstruction to drainage. In this case, the middle ear was draining freely, not only through the mastoid opening, but also through the external auditory meatus, but in spite of this, there was an invasion of the labyrinth. Whether this invasion took place through the oval window, and the carious area in the horizontal semicircular canal occurred secondarily, or whether in-

vasion took place through the horizontal semicircular canal, can only be a matter of conjecture.

A second point of interest was the relatively short duration of the acute labyrinthine symptoms. At the end of forty-eight hours from the inception of these symptoms, the temperature was normal, and remained so in spite of the fact that the labyrinth was the seat of suppuration. The gradual disappearance of the labyrinthine symptoms, such as vertigo and nystagmus, is also a matter of interest.

When we come to examine the tuning-fork tests, the fact that the tuning-fork held upon the forehead was always referred to the affected side, is a phenomenon rather difficult of explanation. With destruction of the labyrinth, we would naturally expect that the tuning-fork would be referred to the opposite side, and that bone conduction on the affected side would be greatly diminished. My case shows, however, that five or six weeks after the labyrinthine symptoms first appeared, the tuning-fork placed upon the forehead, was still referred to the diseased ear, and that the upper-tone limit was not greatly lowered. Bone conduction, however, on the affected side was slightly diminished. Even after the radical operation, we still find the tuning-fork reaction somewhat confusing. The vibrating-fork placed upon the forehead, in the median line, was still referred to the affected ear. Bone conduction on the affected side was slightly diminished,—not absolutely lost, while the upper-tone limit was only reduced to 4.5 Galton. The elevation of the lower tone-limit would simply be indicative of the middle ear lesion and would have no bearing upon the labyrinthine condition.

It is a question in this case, whether it would have been wiser to have operated when the labyrinthine symptoms first appeared. From the satisfactory outcome of the case, I am inclined to think that I pursued the preferable course. Had I operated at the time of the acute symptoms, I rather think that I might have opened up a fresh avenue of infection in the labyrinth. Had the fever persisted, I should naturally have exposed the horizontal semicircular canal and the inner wall of the middle ear at once, by the radical operation; but in the absence of febrile symptoms, I felt that it was better to wait until the diagnosis of labyrinthine involvement could be definitely established. Had I operated within the first twenty-four hours after the appearance of the symptoms, I should prob-

ably have explored the cerebellum, as the symptoms pointed very strongly to involvement of the cerebellum. Such an exploratory operation would, of course, have been unnecessary, and might have been followed by serious results.

While it is impossible to lay down any definite rule in regard to these cases, I believe that if the temperature does not rise above 101° or 102°, it is well for us to wait for a certain interval, and see if the labyrinthine lesion will not be self-limited. If, however, the temperature continues to rise, it is necessary to operate immediately, as then, the only hope of preventing an extension of the inflammation to the cranial cavity, is by prompt operation. This has been demonstrated by Jansen and others.

I think the rule should be, therefore, that, with acute labyrinthine symptoms and pronounced elevation of temperature, operative interference should be at once instituted. With acute labyrinthine symptoms, however, with little or no febrile movement, it is wiser to wait for a few days at least, before opening the labyrinth.

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XXXI.

BLACK HAIRY TONGUE (LINGUA VILLOSA NIGRA).

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The disease, for which some German authors use the characteristic name "Schwarze Haarzunge" (Black Hairy Tongue), and which consists in the growth of long, dark, hairlike structures on the surface of the tongue, is often thought of only as a curiosity. However, the affection is of some interest, since it sometimes happens that the patient believes the disease is of a cancerous nature, and because it very often puzzles the physician on account of its resistance to treatment. Moreover, the condition certainly is not as rare as is generally considered. The etiology and the pathogenesis of the disease are very obscure. In consequence of this, the disease has been given many different names. Some of these are merely the expressions of the authors regarding the nature of the disease.

In the literature I have found the following names:

The older ones—Lingua nigra or Nigrities linguae; English—Black Tongue or Black Hairy Tongue; German—Schwarze Zunge, or Schwarze Haarzunge; French—Langue noire pileuse or villeuse.

Later ones—Hypertrophie epithéliale piliforme (Féreo[20]), Melanotridria linguae (Augier,[4] Maraval,[58] Mracek,[55]); Pseudomelanosis linguae (Birch-Hirschfeld[8]); Lichenoid (Butlin[17]) and Hyperkeratosis (Brosin[14] and others).

The investigators who have considered the disease of parasitic origin have given it the following names:

Glossitis parasitica (Hofheimer[35]); Keratomycosis (Roth[70]); Glossophytie (Pasquier[68]); Mycosis linguae mucorina nigras, Nigrities mucorina linguae (Ciaglinski and Hewelke[20]) and Mycosis mucorina (Sendziak[59]).

It is easily understood how this redundant nomenclature has caused some embarassment. For instance it is seen that Goodale[32] distinguishes the cases of black tongue which he

describes under the head of "Hyperkeratosis lingualis" from those cases in which moulds are found, to which he gives the name "Lingua nigra."

The best designation, in my opinion, is the name that corresponds to the German "Schwarze Haarzunge" and the English "black hairy tongue," viz., "Lingua villosa nigra." This name has the advantage of including the two most characteristic features of the disease: viz., the dark color and the hairy appearance of the tongue.

The first to observe the condition was (according to Arnold) Amatus Lusitanus[51] who, in 1557, saw on the tongue of a man "hairs which came again after they had been pulled out." Later Rayer,[71] in his "Traite des maladies de la peau," 2nd edition, 1835, writes that sometimes there is seen a black color on the back of the tongue, and he names the phenomenon "Negrities." In the same year an anonymous correspondent in the "*Boston Medical and Surgical Journal*"[10] states that his own tongue is black. In 1852 the disease is mentioned by Hyde Salter[92] in Todd's "Cyclopedia of Anatomy and Physiology" and in 1853 Eulenberg[27] wrote a paper entitled "Ein schwarzen Zungenbelag" in which he speaks of the case of a child whose tongue was black far down into the throat. It is doubtful if this was a typical lingua villosa nigra. The same may be said of four cases which Bertrand de St. Germain[91] describes in the year 1855. This author is of the opinion that the black color is due to a pigmentation of the same kind as is found in the skin of the negro. He does not mention the hairy growth which is characteristic of the disease. In 1869 Raynaud[73] published his classical paper "Une nouvelle affection parasitaire de la langue." In this we find for the first time a description of the microscopic appearance of the disease. In the same year, Gubler[33] gave a description of the disease in the "Dictionnaire encyclopédique des sciences médicales." Since that time, the publications have become quite numerous. Several theses concerning this subject have appeared, among which are those of Laveau[46] (1875), Dessois[25] (1878), Ch. Rayer[72] (1883), and Boucher[11] (1903).

I have found in literature 88 different authors (their names may be found in the bibliography) who have written papers on black tongue, and there are probably many more who have escaped my notice. In all, I have found published 128 cases of black tongue, to which I can add ten cases that I have

treated in the Dispensary for Ear and Throat Diseases in the Municipal Hospital of Copenhagen.

The following is the result partly of the investigations of the ten cases examined by me, and partly of the cases in literature.

THE APPEARANCE OF BLACK TONGUE.

The typical black tongue presents an appearance as follows: On the back of the tongue of an otherwise healthy individual is found a brownish-black spot. The spot is located just in front of the circumvallate papillae and extends forward to about one inch from the tip of the tongue. The spot is symmetrically arranged on each side of the median line and the borders and tip of the tongue are covered with a whitish fur. The spot owes its appearance to long hairs, that is, the prolonged papillae filiformes. These have a length of ¼ to ½ inch and are longest in the posterior part and on both sides of the sulcus medianus linguae. In this way the middle part of the tongue may appear very deep, as though there was a defect there. The surface of the tongue can well be compared to the appearance of a wet, long, black-haired dog, or to a wet silk hat. The hairs are directed from behind forward and if the tongue is rubbed in the opposite direction the appearance just mentioned is noticed. The surface of the tongue is somewhat slimy and on the base and in some places between the papillae vallatae, there is a whitish fur that resembles thrush. When the black spot is scraped with a curette the prolonged papillae are very easily cut off and do not leave a bleeding surface. These papillae are conical in shape and are black only at the apex of the cone. At the base they are thick and gray. They float in an opaque, mucous fluid.

In black tongue, there are these two characteristics to be found, viz., (I) more or less hypertrophy of the papillae filiformes, and (II) the dark color of the papillae. Most of the cases in literature show these two peculiarities and can be grouped under the head of lingua villosa nigra. All cases which do not show these marks should not be given this name. Consequently, the cases described by Curtis,[23] Krimer,[40] Eulenberg,[27] and Bertrand de St. Germain[9] cannot be regarded as genuine linguae villosae nigrae. Naturally there may be some difference in the appearance of the cases within the said limits.

We may at times find some difference in the nature of the color. Although the color commonly is black or brownish-black, a yellowish-brown color has been found. In one case, the tongue had a dark greenish-gray appearance, while in another the spot was black in the center and green on the borders. In still another there was a white center with a dark circumference. Sometimes, it has been noticed that the color changes. In one case of Rydygier's[81] the tongue was at first black, but later in the disease turned blue. In the case described by Mickulicz and Michelson[59] the tongue was at first blue but later when the stain had disappeared it presented a whitish, furred appearance due to the long papillae. Yet, in all these cases the results of the microscopic examinations were similar. It is therefore probable that there has existed an analogous condition in all of these cases.

The form, position and size of the colored spot may vary. As a rule the spot is symmetrically arranged along the median line of the tongue just in front of the papillae vallatae, but it may be found on one side or at the tip of the tongue. It may be as small as a one-cent piece or it may extend over the entire surface of the tongue from the tip to the circumvallate papillae and to the borders on either side.

SYMPTOMS OF BLACK TONGUE.

In many cases, the disease gives no symptoms and is discovered accidently by the patient or the physician. In other cases, many disagreeable and severe symptoms are present. In my opinion, there are many cases in which the black tongue is not responsible for these symptoms, namely, those cases in which the patient besides having this disease has suffered from a more serious trouble which latter may be the cause of the symptoms. Only in those cases of black tongue affecting otherwise healthy individuals and where the symptoms appear and disappear with the disease, can one safely say that these symptoms are due to the disease. Considering these facts, it cannot be said that one severe symptom is actually due to the black tongue. The symptoms following this disease are all local: dys- and paresthesia of the tongue; sensation of dryness, tickling, etc., in the mouth and throat; sometimes pain in the tongue or the gums; in some cases the sense of taste is weakened, while in others it is increased to such an

extent that some foods produced the sensation of pain. In some patients the disease produces fetor ex ore.

COURSE OF BLACK TONGUE.

The disease usually develops in the following manner: A white spot first appears. This spot is due to the prolonged, hypertrophied papillae filiformes. It subsequently is stained a grayish-brown and later becomes black or brownish-black. This sometimes takes place in a very short time, sometimes over night (Schmiegelow,[86] Ciaglinski and Hewelke[20]). In one of my cases, the development of the black color covered a period of about four days. In some cases the course of the disease is very short, the color disappearing in from two to seven days. In others, however, the course is more chronic and may extend over a period of several months or years. In one case reported by Sell[87] the duration of the disease was fifteen years. Often, perhaps in all chronic cases, the disease is intermittent, appearing and disappearing at intervals. Out of 48 cases reported in the literature in which the duration is mentioned, 31 lasted one year or more, while the remaining 17 lasted from two days to three months. The most interesting fact in regard to the course of the disease is the relation that exists between the black tongue and the "principal" disease of the patient. The acute cases of black tongue are found on patients suffering from some acute affection (acute gastro-enteritis, pleuritis, etc.), while the chronic cases commonly occur in patients who have some chronic disease (tabes, chronic constipation, etc.). Black tongue is only a symptom that in certain conditions accompanies different acute and chronic diseases.

PATHOLOGIC ANATOMY OF BLACK TONGUE.

Former investigators simply curetted off the papillae and, after putting them in potash, glycerine or acetic acid, and treating with methylene-blue or Bismark-brown, have examined them microscopically. I have examined specimens from five patients, treated by me, in the following manner: The papillae were curetted off from the tongue, hardened in alcohol, imbedded in paraffin, cut on the microtome and examined both unstained and after staining by the Van Giesen method. The advantage of this method of staining lies in the fact that

the keratosed cells are stained a reddish-yellow and the epithelial cells a bluish color. I have also used the Ziem-Nielsen tubercle stain and have been able in this way to bring out the horn cells. By this latter method, the keratosed cells are stained red while the ordinary epithelial cells are deprived of their color by the sulphuric acid. These, however, can be stained later by using methylene-blue. To show the keratohyalin and eleidin granules. I used the v. Ebbinghaus method and the haematein coloring (Unna). In staining the microbes, I used partly the Gram and partly the Weigert method. In all the examined cases the microscopic appearance was about the same. The following is a report of the microscopic examination of papillae from the tongue of a man 70 years of age, who was suffering from progressive bulbar paralysis and an intermittent black tongue: The papillae filiformes are very long (some of them three-fourths of an inch), the most of them being very thin and hair-shaped, while the others are thicker and conical. The epithelial cells on the surface of the papillae are arranged like tiles on a roof. They are hair-like and are loosened at their proximal ends. On this account, they resemble an inverted ear of corn. Commonly, two papillae are attached to the same base, and are connected by hair-like keratosed epithelial cells. The center of the papilla is made up of polygonal epithelial cells, which are stained bluish by the Van Giesen method. The cells have a well-stained nucleus. The surface and tip of the papilla is made up of a closely striped layer of cells which stain a reddish-brown with the Van Giesen method. These are pointed, spindle-shaped keratosed cells which have more or less degenerated nuclei. It is the most superficial cells of this layer which, being loosened at their proximal ends, and projecting out from the papilla, give it its corn-like appearance. Keratohyalin and eleiden granules are not to be found. In the unstained specimens it can be seen that the dark color of the papilla is due to the epithelial cells which have a diffuse brownish appearance. This color is darkest at the tip. Between the papillae can be seen numerous free epithelial cells, some of which are keratosed and have a faintly stained nucleus and reddish-yellow protoplasm (Van Giesen) while others have a pink protoplasm and a distinct nucleus.

On the surface of the papillae, numerous microbes can be found, most of which are round or slightly pear-shaped clear

bodies with a high refractive power. They are all about the size of the nuclei of the epithelial cells, and are arranged in irregular layers both on the surface of the papillae, where they are attached to the barb-like cells, and at the center of the papillae between the epithelial cells. They are readily stained by the Weigert and the Van Giesen methods and are probably spores of mould. Many slightly curved bacilli, some diplococci and some coccobacilli are found, but no leptothrix.

As was said, the papillae in all cases showed similar microscopic appearances. The variations mostly were in the length and thickness of the papillae and the kind and number of the microbes. The large, round, clear microorganisms mentioned above were found in only one case. Bacteria, varying in numbers, were always found. The number depends in a great part on the surface of the papillae. Where there are many, long, barb-like epithelial cells on the surface, many microbes are to be found. Where the epithelial cells lie close together, few microbes are found. The papillae in all cases were connected at their bases. It is therefore not true, what some authors (Mourek,[62] Brosin[14]) maintain, that in black tongue the papillae are always bound together in thick conical bundles.

In all the cases which I treated, I tried to cultivate mould from the tongue. The papillae were scraped off with a sterilized curette, and with a sterile glass tube I rubbed some on a mixture of wheat-bread with 10 per cent gelatine and on malt agar. Some of these were put in an incubator at 24° C. and the others at 37° C. In all cases, colonies of white and gray microorganisms developed but no black or brown moulds.

ETIOLOGY OF BLACK TONGUE.

1. Sex. In the 128 cases of black tongue reported in the literature and the 10 of my own, making 138 in all, 12 were in children below 15 years of age, 56 were in men and 24 were in women. The reports of the other 46 patients were not at my disposal. From this it is seen that the disease has occurred in twice as many men as women. This is no doubt due to the etiologic importance of tobacco (see below).

2. Age. The ages of only 79 patients were at my disposal. These I give in the table below:

Age	Men	Women	Sex Unknown	Total
10 to 14 years of age..	4	3	2	9
15 to 19 years of age..	1	1	..	2
20 to 29 years of age..	13	4	..	17
30 to 39 years of age..	7	2	1	10
40 to 49 years of age..	9	2	..	11
50 to 59 years of age..	6	1	..	7
60 to 69 years of age..	5	3	..	8
Over 70 years of age..	7	8	..	15
Total	52	24	3	79

(Among those numbered under "over 70 years of age" are the ones mentioned in literature as "old.")

From the above table it is clear that the assertion of most authors that black tongue is found almost exclusively in old individuals does not hold good. While there are 30 patients of more than 50 years of age, there are nine cases in children and 40 in persons between 15 and 50 years of age, and among these last there are 17 whose ages range from 20 to 30 years.

3. Different local and general diseases as etiologic factors: In 13 of the reported cases the patient was otherwise perfectly healthy. Two of these were children. In all the other cases, the patient was suffering from some other severe disease. Twelve of the patients had syphilis and of these 2 were suffering from stomatitis, 3 had papules in the mouth and 2 were excessive tobacco smokers. Seventeen patients had different affections of the mouth, pharynx or larynx (of these only 1 was more than 70 years of age, the others were less than 50). Nineteen patients had various affections of the stomach or intestines. Twenty patients were in a state of great weakness either on account of some severe acute trouble (scarlatina, typhoid fever, influenza, etc.), or of some grave chronic disease (various nervous diseases, tuberculosis, etc.), or of old

age. Other intercurrent diseases reported were: Nervousness, 3 cases; asthma, 1 case; pleuritis, 1 case, emphysema pulmonum, 1 case; epilepsy, 1 case; stricture urethrae, 1 case, and diabetes. Of these 8 patients, 5 were more than 60 years of age.

4. The more intimate etiologic factors and the pathogenesis: During the past years many theories have arisen as to the origin of black tongue. Hutchinson[87] maintains that all cases are due to an illusion or to an accidental coloring of the tongue. The other authors may be divided into two groups, in regard to their opinions concerning the etiology of the disease. One group claims that the disease is due to microorganisms while the other maintains that these do not play a part in the development of the affection. There are also differences of opinion among those of the first group regarding the pathogenesis of the disease. (a) Some say that black tongue develops from a black mould which grows on the surface of the tongue. (b) Others have the opinion that microorganisms first produce an hypertrophy of the papillae and then produce the black color. (c) A third group believes that microorganisms produce an hypertrophy of the papillae but do not produce the stain. (d) The last group maintain that the black color is due to the microbes but that the hypertrophy is not.

(a) Ciaglinski and Hewelke[20] were the first who were of the opinion that black tongue was caused by the growth on the surface of the tongue of black moulds and, later on, Schmiegelow[86] and Sendziak[89] were of the same opinion. In the year 1893, the two first named investigators succeeded in cultivating a black mould from the surface of a black tongue which had developed acutely in a patient suffering with influenza. The mould grew at 15° to 25° C. on potatoes, bread, and particularly well on wheat bread with 5/10 per cent gelatine. At 37° C., it could not be cultivated. The growth appeared as an umbrella-shaped columella on which developed spores, which were first white and later became black. Ciaglinski and Hewelke called the mould Mucor niger. The next year (1894) Sendziak[89] cultivated a similar fungus on wheat bread gelatine; it was taken from a black tongue of the usual typical appearance. However, the growth differed from that described by Ciaglinski and Hewelke in two respects: (a) it did not grow on potatoes, and (b) it had an oval, not an umbrella-shaped, columella. From another case,

one of brown tongue, Sendziak[89] cultivated a similar mould, whose spores were brown or yellow. Later (1905) Sendziak[89] states that Paltauf in Vienna found in a patient suffering with black tongue and peritonsillar abscess (also, icterus, enteritis, and fever) Mucor corymbifer both on the tongue and in the pus from the abscess.

Schmiegelow[86] has cultivated moulds from two cases of black tongue. The first case was a chronic black spot on the tongue of a man 42 years old. The microscopic appearance was of the usual nature, hypertrophied papillae filiformes, etc. He found, however, in this case two different types of mould: (a) a chocolate-brown hyphomyceta resembling Oospora ruberrima sacc. and (b) a dark mould, Trichosporium chartaceum (Pers.) sacc. which he (Schmiegelow) regards as pathogenic. The second case was an acute black tongue which developed over night in a lady 70 years of age who was suffering with stomatitis and acute gastro-enteritis. The black spot was situated on the front part of the tongue and had the usual microscopic appearance. Cultures on wheat-bread-gelatine after a week produced colonies of a brown color made up of the mould, Hormodendron cladosporides. Schmiegelow thus concludes that black tongue is produced by the growth of a black mould on the surface of the tongue and he maintains that the reason that other investigators have not been able to find this mould is that they have not used the proper cultivating medium, viz., wheat-bread-gelatine.

All other investigators who believe in the parasitic theory recognize that black tongue depends upon (1) an hypertrophy of the papillae filiformes and (2) upon the black stain of these. Some of these are of the opinion that both the hypertrophy and the black color are produced by parasites. Among these is Maurice.

Raynaud,[73] (1869) who was the first to find microorganisms in black tongue and who was the first to give a careful description of the microscopic pathology of the affection, found (in two out of four cases) on the surface of the papillae, large quantities of round or oval bodies with high refracting power varying from 3/5 micra in size. Though he did not find any mycelia, he considers the bodies to be spores similar to trichophyton, and holds them responsible for the affection.

In 1878 Dessois[25] in his thesis describes similar bodies. He had examined three cases of black tongue. In the first case

he found small bodies of the same size as those Raynaud described but they were arranged in straight lines vertical to the surface and not in irregular heaps as were Raynaud's spores. In the second case he found in some places small heaps of spores which were somewhat larger than those in the first case. In the third case he found spores in isolated groups on the hairs and having an average size between those of the first and second case. In spite of the different microscopic appearances and although autoinoculation gave a negative result, Dessois believed that he had found the specific microbe of black tongue and called it glossophyton. He believed that the microbes located on the base of the papillae, by their irritation stimulated the growth of the papillae and that later they produced the stain.

Schech,[83] in his text-book of 1885, joins those believing in the parasitic theory, based on the publication of Dessois. Two years later, however, he had the opportunity to examine a case of black tongue but did not find any spores and consequently abandoned the parasitic theory, which he opposed in the next edition of his text-book[84] in 1896.

Lannois[45] (1888) was of almost the same opinion as Dessois. In two cases of black tongue he did not find any microbes but in a third case he found round or oval colorless refracting bodies which looked like spores and which did not stain with the analine dyes. Attempts to cultivate them were unsuccessful. Lannois maintains that a local disturbance of nutrition prepares the ground after which the bacteria become the decisive cause of the development of the hypertrophy and the coloring of the papillae.

(c) Some authors maintain that the hypertrophy but not the coloring is produced by parasites. This theory was brought forth in 1887 by Roth.[79] He describes two patients whose tongues were covered with hypertrophied papillae filiformes. In the first case the color was white while in the second it was black, the tips of the papillae being stained (by tobacco). The author did not succeed in making a pure culture of the microbes found, among which were sarcina, oidium albicans and leptothrix. Roth believes that the hairy structures are produced by the usual microorganisms of the mouth and names the condition keratomycosis.

David[24] assumes that the cause of the hypertrophy is the irritation of the bacteria which are located at the base of the

papillae and that later they grow and cover the latter as a cloak. Rostowzew[73] is of this opinion also and on cultivation found a cladothrix and a sort of yeast. Inoculation of man and animals was unsuccessful.

(d) There are still some who believe that the color but not the hypertrophy is produced by parasites. Most of these have not examined cases personally but rely on the investigations of others.

Cornil and Ranvier[22] are of the opinion that the black color is produced by myriads of black micrococci. These they believe to be the same as are usually found in the mouth, differing only in color. These are arranged in zoogloea as numerous small round immobile bodies and are intensely stained with the analine dyes.

Butlin[18] attributes the hypertrophy to the fact that the friction of tongue is less in front of the papillae vallatae and he believes the color to be due to microorganisms.

It seems as if Vignal had the same opinion. He relates that Malassez has cultivated the same spores as Dessois[25] found, and that he had further found similar spores in 18 cases of "white tongue." Dessois assumes that the black and the white tongue are produced by two varieties of the same microbe, a white and a black one. These microbes which Dessois named glossophyton are in Vignal's opinion identical with the alphacoccus described by him.

Finally there are several authors who believe the affection to be of parasitic origin but do not mention anything particularly about the pathogenesis. Hofheimer[35] names the disease glossitis parasitica. Papon[66] suggests that the cause of the reappearance of the disease is the resistance of the parasites. Gaston and Nicolau[31] believe that black tongue is produced by a variety of leptothrix and Boecher[16] also is inclined to consider the parasite as being a leptothrix in the early stage of development. Pasquin[68] found the spores described by Dessois. Leviseur[49] is of the opinion that the dark color of the papillae is produced by (1) drying, (2) chemical influence of food, (3) certain bacteria which act as chemical agents, and (4) certain drugs. He found sulphur but no iron in the black stain and considers it identical with the color in the keratosed cells (Unna[110]).

(3) Those who do not believe in the theory that microbes are etiologic factors in black tongue have many different opin-

ions concerning the etiology and pathogenesis of the disease.

Armaingaud[2] believes the cause is a disturbance of the vasomotor nerves as is seen in the chromidrosis of the skin.

Solis Cohen,[21] Chevalier,[19] Mourek[62] and Masoin[54] maintain that black tongue is due to trophic disturbance, and the last named compares the affection to papilloma.

Hyde Salter[82] enunciated the theory that black tongue is due to pigment granules which are identical to those found in the scrotum at the age of puberty, when the skin is filled with blood. Matthieu[56] assumes that "des granulations graisseuses dans les gaines épithéliales" contribute to the dark color and finally Goodale[32] found on the surface of the papillae refractant granules of a yellowish-brown color together with characteristic bacterium (probably large cocci or spores). It is interesting from an historical standpoint to note that Rayer[71] (1835) mentions in one form of black tongue a "coloration pigmentaire" by which he distinguishes it from the artificial form due to food and drugs.

It is only by a few, and especialy the older authors, that pigment granules are reported to have been found in cases of black tongue, so that we can discard the theory which claims all cases of the disease to be due to pigment granulae. Wallerand[96] claims that these observations were wrong.

Most of the investigators during the last few years are of the opinion that black tongue is due to a hyperkeratosis of the epithelium of the papillae filiformes. This theory owes its origin to Schech[85] (1887). He found that the papillae cut from the tongue were completely keratosed and he maintains that the affection is due to an hypertrophy of the papillae filiformes with keratinization and pigmentation. Brosin[14] is also of this opinion. He declares that the affection is due to a genuine hyperkeratosis connected with a consecutive degeneration atrophy (brown coloring) of the cells. Others that join in this theory are Dinkler,[26] who compares the formation of the pigment in the horn-cells of the papillae with the pigmentation of the horn-pearls in epithelioma; Wallerand[96] who compares black tongue with psoriasis linguae, and Mracek,[63] who considers the affection to have the same pathogenesis as pachydemia laryngis. Vollmer,[102] Maraval,[53] Johnston,[38] and Angier[4] also consider black tongue a hyperkeratosis. Only Maraval of all the investigators found fine granules in the epithelial cells. These granules were stained pink

with hematoxylin and were also stained by the Gram's method. He considered them to be eleidin.

We must base our opinion as to the true pathogenesis of black tongue on a consideration of all the reported cases and on the different theories as advanced by the different investigators.

First let us consider the parasitic theory: This theory may be supported by different means—(1) microscopic examination and cultivation of the microbes; (2) inoculation; (3) cases of infection.

By microscopic examination and cultures, sometimes numerous bacteria are found. These are for the most part the same as are found in healthy mouths (David[24] found in normal mouths 19 different kinds of bacteria and Mills[108] found 25). Among others the following microorganisms have been found in cases of black tongue: leptothrix, cladothrix, yeast, sarcina, oidium albicans, jodococcus vaginalis Miller, staphylococcus pyogenes aureus and albus, spirochaeta bucallis, bacillus subtilis, and glossophyton Dessois. In some cases none or very few are to be found (Lannois,[45] Dessois,[25] Schech[85]).

The distribution of the bacteria is irregular in most cases. Some papillae may be entirely covered with microorganisms while others have only a very few, the number of bacteria having no relation to the extent and severity of the affection. These facts do not support the parasitic theory. Up to this time no one has succeeded in finding a parasite which is present in all cases of black tongue. Only a few of the reported bacteria can be considered; first, the dark moulds cultivated by Ciaglinski and Hewelke[20] (1 case); Schmiegelow[86] (2 cases); Paltauf[90] (1 case), and Sendziak[89] (2 cases). It is a striking fact that in these six cases we have to deal with six different moulds. Further, it is strange that none of the other authors has found moulds. Rydygier,[81] Vidal,[100] Lancereaux[43] and Sell[86] found mycelia, but nobody has been able to cultivate dark moulds from a black tongue, although several investigators (among others Moure[61] and Blegvad in all their cases) have used wheat-bread-gelatine, which should be the best culture medium.

It is a strange fact that the moulds mentioned can only be cultivated at 21° to 25° C., while they grow in the mouth at 37° C. Some explain this by saying that the moulds grow in mouths of patients who breathe through their mouths,

where the temperature is below the normal and the tongue immobile. This explanation can only hold in those cases where the patient uses mouth respiration. In some of Schmiegelow's and Sendziak's patients this was not true. These patients were suffering with a chronic affection while with the others it was an acute affair. Thus there is not a single case reported in literature that is convincing, and one is inclined to believe that the finding of moulds in all these cases was purely accidental. Moulds are present everywhere, as is well known, and it is not probable that they should play any part in the etiology of the disease.

Another parasite which might be considered is the "spores of moulds" which Raynaud[73] has described. Dessois[25] named them glossophyton and Vignal[101] considered them identical with his alphacoccus. Many investigators have found this microbe (Blegvad in one case, Brosin,[14] Lancereaux,[43] Laveau,[46] Lohéac,[50] Masoin,[54] Pasquier,[68] Rayer,[72] Wallerand[96] (in two cases), Weill[98] and Vignal[101]). Many investigators have searched in vain (Blau,[9] Blegvad in 4 cases, Tereol[89] Mourek,[62] Richter,[76] Stocker[93] and Wallerand[96] in one case). This parasite is sometimes found on healthy tongues (Ch. Rayer,[72] Vignal[101]), and Brosin[14] is perhaps right when he says that the parasite is only a badly developed oidium albicans.

Inoculation.—It is evident from what has been said above that microscopic examinations and cultures have not given reliable support to the parasitic theory. In addition, no one has succeeded in inoculating the affection on the tongue of a man or an animal; not even in those cases where the experiments were done under the most favorable circumstances (as for instance by Wallerand[96]) have the investigations given a positive result. Wallerand tried to inoculate both papillae and cocci from a culture taken from a case of black tongue, on to the tongue of a patient with phthisis, in the last stage of the disease. Rostowzew[78] made an attempt on animals and man, Mourek[62] on man, Masoin[57] on animals, Dessois,[25] Gundobin[84] and Rayer[72] on themselves. In all cases the results were negative.

Infection.—It could be expected that black tongue, supposing it to be of bacterial origin, would sometimes be carried from one person to another by infection. Rostowzew[78] is the only author who mentions a case that could be explained in

this way. He reports the case of a man and his wife who were both affected with black tongue, but this could be due just as well to the fact that both eat the same food. These facts I think prove that there is no ground for the parasitic theory.

Among the remaining theories there is only one that may be brought forward and that is the theory of Schech that black tongue is due to an hyperkeratosis of the epithelium of the filiform papillae. The proof of this theory can be gained only by microscopic examination.

It is remarkable that most of the investigators have been satisfied with making only an examination of the fresh papillae, treated with acetic acid or potassium hydroxid and staining with methylene blue, Bismark brown, or similar stains. I feel sure that the results would have been different if they had examined double-stained paraffin- or colloidin-sections. In specimens made in this way, the superficial cells are keratosed and are either without a nucleus or with a very faintly stained one. The long thin papillae consist exclusively of such cells firmly bound together. But when specimens from normal or furred tongues are examined in the same way, it is found that aside from the length of the papillae there is no essential difference between these papillae and those from a case of black tongue. I have examined microscopically curetted papillae from five normal and two furred tongues, and from one tongue (postmortem) where the tongue had been hardened by 10 per cent formalin. In all the sections numerous papillae filiformes were found, smaller than those from black tongue but otherwise not differing essentially. The superficial cells were keratosed to the same extent, had no nuclei, became orange after v. Giesen and red after Ziehl-Nielsen. The specimens differed mostly with regard to the bacteria. In some of the sections the papillae were covered with a thick layer of leptothrix (short, thick bacilli arranged in chains, Gram-positive), while in others only a few or no leptothrix were present, but large masses of zooglea consisting of bacilli and cocci. It is thus evident that the papillae from normal tongues on an average are just as much keratosed as those from black tongues and we may assume that the intense keratinization cannot be the only cause of the color in this disease. In one of the acute cases which I treated the black tongue developed in the following manner: A white spot first appeared extending from the elongated papillae filiformes and in the course of three or

four days it became gray, then brown and finally black. The disease spread slowly to the different parts of the tongue so that it was possible to obtain specimens from white and black areas at the same time. I made sections of the two and found them to be the same microscopically. The keratinization, bacteria, etc., were alike except that in the unstained specimens the papillae from the dark spot were somewhat darker in color than those from the white area.

As a result of the above investigation I feel safe in saying that it is not yet proven that black tongue is due to an excessive keratinization and pigmentation of the epithelial cells of the papillae filiformes. The superficial cells of the papillae of the tongue including the papillae valatae, are always keratosed and there is no reason to assume that they should be more so in black tongue than in a normal tongue. The degeneration of the horn-color, which is mentioned by some authors, is also only hypothetic and its existence should be proven by the following facts: (1) The black color of the horn-pearls in epithelioma is due to a degeneration of the horn-color, and (2) the color in black tongue is insoluble in water. The latter fact may be due to a penetration of the epithelial cells by a color from without which cannot be washed out again, as in the staining of horn-cells with picric acid.

It is apparent that none of the foregoing theories are capable of standing a thorough criticism and that we must find a new explanation for the etiology and pathogenesis of black tongue.

In my opinion the disease originates in those persons with normally well developed papillae filiformes which have for some reason become prolonged and then have become stained by some chemical process from without. The prolongation of the papillae may be caused in different ways. It may be due to some form of irritation as smoking, to the elimination through the mouth of certain drugs as mercury, or as is oftenest the case to a slight glossitis brought about by an affection of the mouth, nose, throat or intestinal tract. The hypertrophy may be due to a nutritive disturbance or to mouth-breathing, especially in old and sick persons. Perhaps bacteria, too, may play a part. It is difficult to say whether or not black tongue is accompanied in all cases by a stomatitis as the microscopic examinations which have been made were made on papillae which were simply curetted off from the tongue. Goodale[32]

says that in the corium he found an infiltration of small round cells with a protoplasmic reticulum, and I found in one case some leucocytes in the base of the papillae.

The part that the acidity of the saliva plays in the hypertrophy of the papillae is not clear. Some authors attribute great importance to this factor. In my opinion it can just as well be a matter of secondary importance due to the same cases as black tongue: immobility of the tongue, indigestion, stomatitis with fermentation and subsequent formation of acid.

The coloring of the prolonged papillae is due first of all to food (and wine); also tobacco (especially chewing) and drugs (iron, mercury, etc.). Probably mouth-breathing also has some influence since the drying of the surface of the tongue may produce a dark color.

This theory has already been partly advanced by Gallois[30] (1869) and Rydygier[81] (1891) and it is supported by several facts. The different cases of black tongue have different colors and it has been often found that different colors follow one another in the same patient at different times (black, blue, brown) (Rydygier,[81] Mickulicz[59]). Also black tongue may change to a white tongue with long, uncolored papillae.

A factor in favor of this theory is the importance of tobacco in the development of black tongue. Most authors do not mention the use of tobacco and in the cases reported by Brydon,[15] Lake,[42] Masoin[54] and Master[55] tobacco was not used at all. However, 16 of the reported cases of black tongue were in patients who smoked tobacco and of these 10 were very great smokers. In 6 of these patients no other disease was mentioned and we are justified in assuming that they were otherwise healthy.

The other 10 suffered with different diseases. Four had syphilis, 2 had angina, 1 had enteritis, and 1 colica saturnina. The importance of tobacco in the etiology of black tongue is evident in Maraval's[53] case. In a man, 42 years of age, the disease disappeared as soon as the patient abandoned the use of tobacco, but reappeared with all the characteristic symptoms when the patient, two months later again began the use of the tobacco. Among the 73 adults with black tongue, there were 16, or 22 per cent who were smokers, and 30 per cent of the 45 men also smoked tobacco. These figures are probably too small as few authors mention the use of or abstinence from tobacco in their patients.

The influence of drugs is to be noted in the following cases: Dinkler,[26] in case No. 3, reports a patient who had white tongue which appeared and disappeared and finally developed into a black tongue when the patient took iron-powder. The scraped-off papillae gave a reaction with ferrocyanid of potassium and included a granulated pigment. Blair[9] (case No. 4): woman, 21 years of age, with sinusitis maxillaris operated through the alveolus; first, grayish-yellow hairs were seen on the tongue and two days later these hairs were colored black through the use of iodoform gauze. They disappeared spontaneously. Balfour-Graham[5] reports a case of black tongue, in a patient suffering with caries of the first dorsal vertebra, which developed while he was taking large doses of mercury. It seems that at the same time he had a severe stomatitis, for all of his teeth fell out.

Cases are also reported where the disease has developed during the use of opium (Hofheimer[35]) and bismuth (one of our cases). It can be easily understood that black tongue may develop after the application on the surface of the tongue of chromic acid (Leviseur,[49] two of our cases) or silver nitrate (Schmiegelow[86]). The importance of coloring matter from without is evident from the following case, cured by us. The patient, a man, 21 years of age, was suffering from a violent attack of mercurial stomatitis during which he developed a case of black tongue. The black color was seen not only on the tongue but also on the teeth which were entirely covered with a thick, shiny, black coating. This coating was scraped off and examined chemically. It consisted of organic substances, potassium and sodium but did not contain any mercury. This goes to prove that mercury does not act in this way, since black mercuric salts (viz., mercuric sulphide) are found in the mouth. It is probable that the mercury by its excretion irritates and thus produces a stomatitis on the basis of which the black tongue develops. When black tongue is so often found in persons suffering from syphilis it is probably because syphilis is so frequently accompanied by affections of the mouth.

There is, in my opinion, no difference between a black tongue and the ordinary furred tongue. Müller[109] and Fuchs[108] emphasize the fact that there are two different kinds of furred tongues. The genuine fur consists of loosened epithelium, leucocytes, moulds, residues of food, etc. The "not genuine"

fur is made up of the hair-like prolonged papillae filiformes with their keratosed epithelial cells. Naturally it is the latter kind of furred tongue that the black tongue resembles.

The location of the black spot just in front of the papillae vallatae is explained by the fact that the papillae filiformes normally are most developed at this place (Koelliker[106]) and that the friction against the teeth and palate is less here.

Black tongue sometimes has a very acute course and occasionally lasts for years. This will be understood when we bear in mind that the etiologic factors (the nutritive irritation and the coloring matter) sometimes are present for a long time and sometimes for only a very short time.

The fact that the color has its seat especially in the uppermost parts of the papillae depends upon the fact that the food in passing as well as the drugs, etc., only touch the tips of the papillae.

DIAGNOSIS.

In making the diagnosis lingua villosa nigra, we need only consider the two principal symptoms: (1) The hypertrophy of the papillae filiformes and (2) the dark color. Not every dark stained tongue, however, can be called a black tongue. For instance, the normal tongue may be temporarily colored by certain coloring matters passing through the mouth. The tongue is colored black by ink, red wine, mulberries, and certain kinds of cherries; it is colored brown by licorice, fresh nuts and dried plums, and brownish-gray by chocolate. Tannin preceded by iron also stains the tongue black. Black tongue cannot easily be confounded with leucoplakia oris (psoriasis linguae) because in the latter the spots of thickened epithelium are either white or gray, but in persons who smoke tobacco to excess, and such persons are more likely to be affected with leucoplakia, the epithelial hypertrophy may be grayish-black. The surface of the spots is perfectly smooth and the papillae do not show any hypertrophy. Moreover, the spots most commonly are found on the borders and the tip of the tongue and inside the cheeks. Accompanying the morbus Addisonii, one often finds in the mouth the so-called melanoplakia, dark spots due to the deposit of pigment in the rete malpighi, without proliferation of the epithelium. These spots, however, appear mostly on the lips and inside the cheeks. Patients who suffer with tuberculosis of the lungs

often show signs of melanosis, especially in the face. The spots begin to make their appearance on the nose or forehead and seldom spread to the mucous membrane. If this does occur the affection must be distinguished from lingua villosa nigra which also, as before stated, ocassionally is found in tuberculosis patients.

TREATMENT.

Since black tongue is not dangerous to life and since it very often disappears spontaneously, it needs treatment only in cases where it is accompanied by disagreeable symptoms, viz., pain, disturbance of taste or the like, or when a neurasthenic or hypochondriac patient asks for treatment.

In times past, a great many different remedies have been proposed and used, a fact which shows that the affection is resistant to most of them. A therapeutic procedure used in very many cases is to scrape off the papillae with a curette or to cut them off with a pair of scissors. This treatment nearly always causes the affection to recur in a few days, since it acts as an irritant which stimulates the growth of the papillae. The use of cauteries such as lactic acid, chromic acid, silver nitrate, trichloracetic acid, etc., shows a similar result, because of which they are unsuitable for treatment. They increase the existing irritation and besides in many cases increase the dark color.

When we consider that black tongue has been thought to be of parasitic origin, it is understood that almost all of the known antiparasitic remedies have been used but always without producing any considerable effect (bichorid of mercury, carbolic acid, boric acid, etc.).

In support of the theory that black tongue is an hyperkeratosis there have been applied horn-dissolving remedies (salicylic acid) but this treatment has also been unsatisfactory, an indirect proof for the untenableness of the hyperkeratosis-theory.

The best remedy is the local application of a 10 per cent solution of hydrogen peroxyd which destroys directly the coloring matter by the influence of its oxygen (which acts in statu nascendi). The remedy must be applied every day and sometimes where the color is very dark two applications daily will be necessary.

As the cause of black tongue is always a slight glossitis or

stomatitis some form of mouth wash should be used (chlorate of potassium, boric acid, permanganate of potassium).

The patient should always be told that the affection is very apt to recur, especially when the causes of the affection (use of tobacco, indigestion) are not removed.

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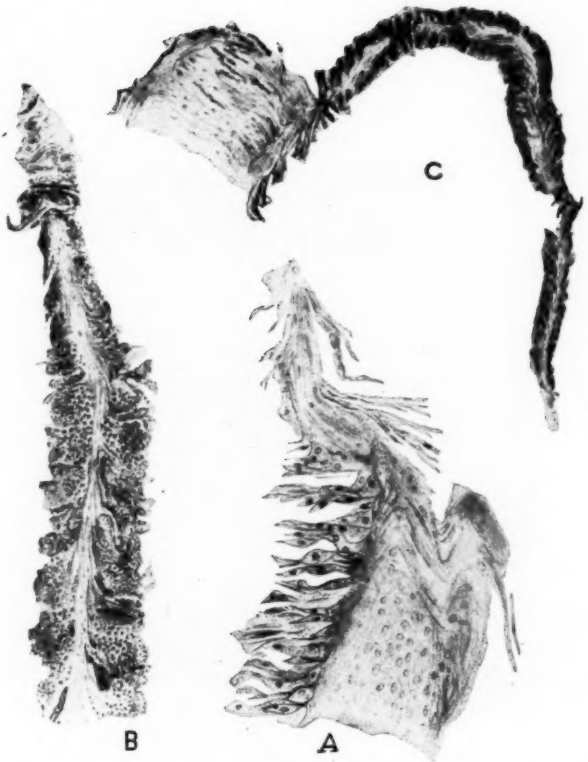

FIGURE 1.

A.—Black Tongue I. Paraffin slide of papilla filiformis. Van Giesen stain. Enlarged 220 times. Tile-shaped epithelial cell on surface.

B.—Black Tongue II. Paraffin slide of papilla filiformis. Van Giesen stain. Enlarged 220 times. Numerous large cocci (Spores(?) Dessois glossophyton) on the surface.

C.—Same specimen as B., 70 times enlarged, showing two papillae connected with epithelial cells at their base.

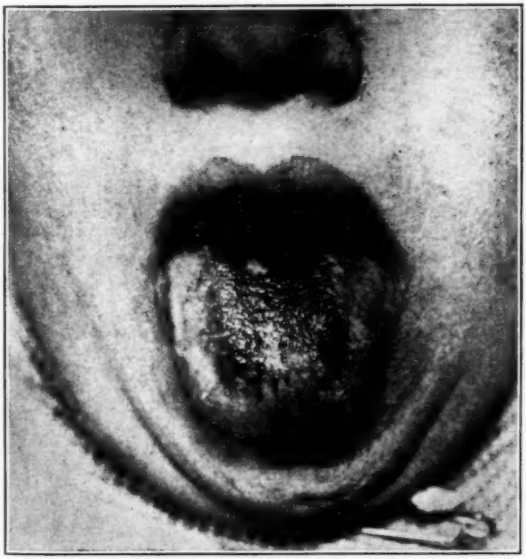

FIGURE 2.

Black Tongue I. Caroline F., aet. 22. Syphilis and severe mercurial stomatitis. After a local application of chromic acid, developed black tongue which lasted for more than a month with intermissions.

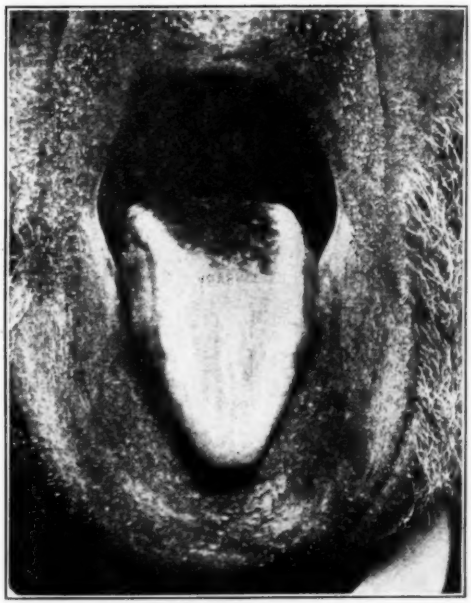

FIGURE 3.

Black Tongue II. Christian C., aet. 70. Progressive bulbar paralysis and a chronic intermittent black tongue.

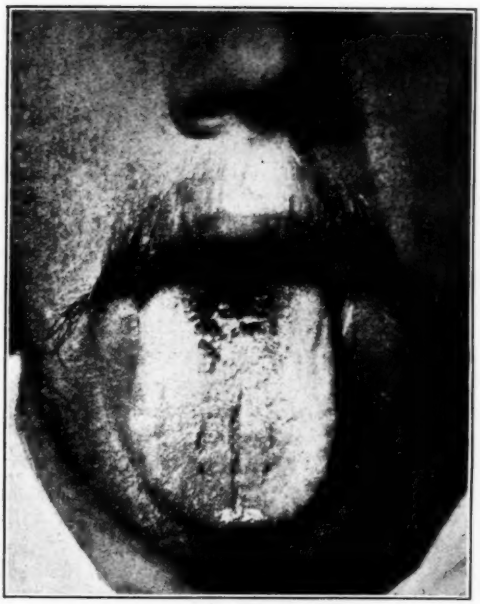

FIGURE 4.

Peter C., aet 23. Syphilis with no stomatitis. Acute case of black tongue.

XXXII.

PAPILLOMA OF THE LARYNX IN CHILDREN; A FURTHER CONSIDERATION OF THE SUBJECT.*

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Three years ago I had the privilege of reading a paper on this subject before this Association. I wish now to report the results in the four cases which were then incomplete, to report four new cases and to review the literature which has appeared in the past three years, and, in the light of more experience and thought, to modify somewhat the statement which I then made in regard to its treatment.

In reporting the subsequent histories of cases 9, 10, 13 and 14 in my first paper, I will begin with a brief resumé of each case as they were then reported.

Case 9.—Boy, 6 years old, when first seen in February, 1901, with symptoms of a year's duration. Tracheotomy for severe dyspnea. Papilloma removed. Eight months later, larynx again filled with growth. Papilloma again removed. In February, 1902, larynx free from growth above vocal cords but large mass below glottis. Nothing done. Seen from time to time, growth diminishing. In October, 1904, only a small growth below glottis, growing from anterior wall. This was the condition of the case when last reported. On June 27, 1905, he was admitted to the hospital and, under ether anesthesia, a small growth was removed from the larynx through the Killian tube spatula and a small mass from the tracheal wall through the tracheal opening. On July 5th, no papilloma was visible in larynx or trachea. September 11th, no papilloma. Tube removed. The patient reported again on March 17, 1906, when the larynx was normal. Under ether anesthesia the fistula left by the tracheotomy tube was dissected out and the wound sewed up. Recovery was uneventful.

*Read at the Congress of the American Laryngological Association in Montreal, May, 1908.

CASE 10.—George W., 9 years old, when first seen in August, 1904, with history of hoarseness for six months and dyspnea for two weeks. Tracheotomy performed. Growth removed in September, 1904, and again in February, 1905. It recurred rapidly and further operative interference was abandoned. Such was the state of the case when I reported it. On May 15, 1905, the record says: Papilloma not increased in size since last visit. Rises from right side and anterior commissure. July 10th: Papilloma appears smaller. August 28th: Growth about the size of half a split pea in anterior commissure. December, 1905: The same. May 11, 1906: Papilloma having remained quiescent for ten months it was decided to remove what remains. The patient was etherized and placed in the Rose position. By means of the tube-spatula a good view of the larynx was obtained. A growth the size of a pea was seen on the anterior tracheal wall below the vocal cords and was removed with alligator forceps and curette. On May 24th, there being no evidence of papilloma anywhere, the tracheotomy tube was removed. It had been worn for one year and nine months. On July 18, 1906, the edges of the tracheal fistula were refreshed by dissecting out the scar tissue and the edges brought together and sutured. July 22, 1907: Larynx free from papilloma, vocal cords normal in appearance, voice good. Tracheotomy wound has healed nicely.

CASE 13.—Nellie S., aged 6, entered the hospital on July 5, 1904, with a large papilloma, completely hiding the glottis. Tracheotomy was done and on September 26th the growth was removed. It recurred rapidly and over a greater area than before the operation. Under these conditions non-interference was decided on. Note on May 5, 1905: Large papilloma visible through tracheotomy wound, apparently attached above. No change in larynx. September 22nd: Comes in with history of having been ill with pneumonia since June 28th. Papilloma is much diminished. None visible through tracheotomy wound. Breathes fairly well with tube closed and can speak aloud. Examination of chest shows dullness and diminished breathing over lower half of right side, front and back, slight dullness at left base behind. X-ray showed opaque shadow from fifth space to base of right lung with less dense shadow at base of left lung. It was thought that there was fluid on right side but a needle was inserted with negative

results. The patient's general condition rapidly improved in the hospital. Her temperature became normal on October 4th and she felt perfectly well. She was discharged relieved on October 12th.

Diagnosis.—Thickened pleura and chronic bronchitis.

On November 10th the papilloma had increased somewhat, and her general condition was poor. Her temperature was 100, pulse 160 and respiration 40. Physical examination of the chest gave practically the same condition as in October. On November 24th, the condition on the right was about as before, but in the left axillary line there was a dull area about 2½ inches in diameter with fine moist rales. Broncho-pneumonia. The patient had a temperature of 102, chilly feelings, poor appetite, pain in back and sides on cough, which was frequent. The temperature dropped to normal on night of the 24th and remained so. The sputum at this time was large in amount, containing a few influenza bacilli, many pneumococci, some in long chains, all extracellular, and many other bacteria but no bacilli of tuberculosis. On November 29th she was discharged "relieved." The diagnosis was croupous pneumonia. She died at home on February 12, 1906. A letter from the family physician, dated June 16, 1906, contains the following: "The condition of Nellie S. during last few weeks was one of exhaustion and pyemia. She took little nourishment, vomited more or less and raised through tube large quantities of fetid pus, as much as four ounces at a coughing spell. She complained of some pain in region of heart during cough. She had not been able to lie down for a number of months. Sputum showed no tubercle bacilli after repeated examinations during last few months although tuberculosis is in her immediate family."

Case 14.—Jennie M. C., age 2 years, was admitted August, 1904, on account of increasing hoarseness and dyspnea. An examination showed the larynx almost filled with papilloma. Tracheotomy was done and in October the growth was thoroughly removed. In February, 1905, the larynx was again full and it was decided not to attempt further removal. On September 8th papilloma was seen presenting at the tracheal opening as well as in the larynx. On January 19, 1906, while changing the tube, the child was nearly asphyxiated by the closing of the tracheal opening by a papilloma. A portion of this growth was removed. On March 1st a small papilloma,

springing from the tracheal wall was removed through the tracheal opening. On May 21st the growth had diminished. On July 9th the remaining growth was removed through a Kierstein autoscope.

February 13, 1907.—Very small papilloma, 1 to 2 mm. in diameter on anterior portion of left vocal cord.

February 18th.—A small papilloma removed with snare through tracheal opening.

April 10th.—Growth on cord nearly disappeared. No recurrence in trachea. Tracheotomy tube left out.

September 25th.—Growth has gone. Left vocal cord slightly reddened at site of growth.

In April, 1908, the larynx being normal in appearance the patient was admitted to the hospital, the tracheal fistula dissected out and the wound sutured.

Since the above cases were first reported there have been four other cases in the Massachusetts Hospital throat clinic which I will report:

CASE 15.—Alphocena M., girl, age 6, was first seen on January 10th, 1905. She had convulsions a year before and lost her speech a few days after. Speaks now only in a whisper. The larynx appeared to be two-thirds full of papilloma. On January 14th a small piece was removed. On February 11th the growth was seen to have an extensive base but no large pieces. There is no record after this and a letter written to the parents on March 11, 1908, was returned stamped by the post office authorities, "Removed, address unknown."

CASE 16.—Herbert H., age 10, came to the clinic on March 14, 1905, with the history of hoarseness and slight cough for a year. On examination a papilloma was seen apparently protruding from the left ventricle and a smaller one in the anterior commissure. On March 21st the Hooper laryngeal curette was passed twice but no growth obtained. On April 13th there was no material change in the size of the papilloma. On May 25th the growth had nearly disappeared and the voice was fairly loud. The patient was written to in March, 1908, and the following reply received: "The boy's throat has been very well since the last visit. He has grown rapidly and Dr. B., our local doctor, thinks he has outgrown the trouble."

CASE 17.—Annie F., age 7, was seen in August, 1906. There was a history of mouth-breathing, choking at night and inspiratory stridor. The patient was aphonic and there

was marked supraclavicular retraction on inspiration which was whistling. An examination showed a large papilloma on the right cord, causing marked obstruction. Tracheotomy was advised. The patient did not return and a letter sent on March 11, 1908, to the address given was returned stamped "unclaimed."

CASE 18.—Charles B., age 3, was seen first on Mary 22, 1907, with the history of having lost his voice eight months before. The laryngeal obstruction was sufficient to cause aphonia and dyspnea. Examination showed a large mass growing from both sides of the larynx. On May 24th I performed tracheotomy and the patient was discharged on the 31st, wearing the tube. After over three months of waiting, the papilloma remaining apparently the same size, the patient was etherized on September 6th and as much as possible of the growth removed with alligator forceps by direct laryngoscopy. On September 30th an examination under ether anesthesia with the tube spatula showed a very small growth remaining on the right side of the larynx. An examination on March 23, 1908, in the same manner, showed the larynx absolutely free from papilloma. The tracheotomy tube was removed and the tracheal fistula will be closed after waiting two or three months to be sure that there is no recurrence.

The outcome in case 14 adds one more death in the group first reported, making five fatal cases out of the ten in whom tracheotomy was done. The fatal termination in this case does not alter my belief that the method pursued was the right one. If the growth had been removed at first without preliminary tracheotomy and there had been no occurrence at the time requiring an emergency tracheotomy, the growth recurred so rapidly and extensively that tracheotomy would have eventually been necessary. If the growth had been removed by laryngo-fissure there is no reason to believe, from the history of similar cases, that there would not have been an equally rapid recurrence. It is obvious, however, that children wearing tracheotomy tubes should have good home care. The parents should be instructed in the care of the tube and in reasonable precautions against catching cold and that measles should be guarded against as particularly dangerous.

It hardly seems necessary to speak of the advantages of a preliminary over an emergency tracheotomy. In the former the site of the incision is carefully chosen and each step of the

operation is done in a thoroughly surgical manner, while in the latter the importance of opening the trachea as soon as possible makes a careful dissection impossible. John Rogers has said something about tracheotomy which I shall quote later.

Cases 15 and 16 illustrate the type of case which gets well without tracheotomy after one removal of the growth. In these cases the papilloma was evidently in a retrogressive condition. In fact case 16 may be said to have recovered without treatment, since the one attempt to remove the growth failed. Although case 15 is incomplete it seems fair to assume from the diminution in the growth in the month between the first and last examination that this papilloma disappeared without further treatment in the same way that those in case 16 did. A bad prognosis in case 17 without treatment seems perfectly reasonable. Case 18 illustrates well a successful result of tracheotomy and the removal of the growth later when it had ceased to increase.

The literature of this subject during the past three years contains some interesting and valuable articles. A. Neubauer reports five cases on which he operated, three after a preliminary tracheotomy and two without. Four of these cases were cured and one died. This case, a girl of two years of age, had treacheotomy for dyspnea. The papilloma was removed two weeks later. After five weeks the child had measles followed by bronchitis and bronchopneumonia to which she succumbed. One case, a girl of 3 years, had had laryngo-fissure a year before, followed in six months by hoarseness and dyspnea, caused by a recurrence of the growth. This patient wore the tube for over two and a half years. The other tracheotomized patient wore the tube a year and a half. A boy, age not mentioned, had a papilloma on the right vocal cord, causing aphonia. This was removed without tracheotomy and three months later the larynx was still free from growth. A girl of three, with hoarseness for a year and dyspnea for two weeks had a papilloma on the left vocal cord, size of a bean. This was removed without tracheotomy and the patient discharged in two weeks relieved. Neither of these cases can be considered complete as in neither has sufficient time elapsed to warrant assurance of non-recurrence. Neubauer removed the papilloma in all these cases with the modified Löri catheter, an instrument with which it would

seem to be impossible to effect a thorough removal of the growth, except by a happy chance.

Koelreutter reports a case in which he thinks the administration of iodid of potash in small doses caused the disappearance of the papilloma when it had recurred once after removal. It does not seem reasonable to draw any conclusions from one case when we know that papilloma sometimes disappears without treatment.

Garel reports two cases operated on through Killian's tube spatula without previous tracheotomy. Both cases exhibited marked dyspnea and tracheotomy instruments were ready. One case was operated on four times in eighteen days and the child discharged five days after the last operation with the larynx free. In the other case there was an attack of suffocation the night after the first operation for which intubation was done. This child was operated on three times in nine days then operative interference was interrupted on account of an attack of rheumatic fever. Neither of these cases are of value statistically because reported too early.

McCreary reported the case of a girl 2½ years old in which intubation gave no relief, in fact the condition was worse. Tracheotomy was done and the child is still wearing the tube.

In discussing this case John Rogers said that a high tracheotomy with a long curved tube was very liable to make a "spur" behind or a stricture if the opening was made to one side. If the wound is medial and not too high the chance of stricture is slight. Everything depends on where the incision is made. This is a strong argument against an emergency tracheotomy. In not one of my cases was there the slightest evidence of a tracheal stricture. Rogers reported that in one case he had opened the larynx and trachea and cut through the hyoid bone, excising all the papillomata and then put in an intubation tube and sewed the wound up tight. He does not mention how long the intubation tube was kept in or the condition of the voice afterwards. This operation had been done nearly a year before and there had been no recurrence. He expressed the opinion that recurrence was probably due to deficiency of excision. It is surprising that he could make such a statement as this on the strength of one case in the light of the literature of this subject.

Logan Turner showed before the Laryngological Society

of London the larynx of a boy, aged 10, who died suddenly while eating, apparently choking to death. He had hoarseness but no history of dyspnea or choking before. Almost the whole larynx above the glottis was filled with papilloma.

Wingrave is inclined to consider laryngeal irritation due to mouth-breathing caused by the presence of adenoids, a factor in pathogeny. He states that papillomata have been reported not only as co-existing with adenoids but as having spontaneously disappeared after the removal of the latter. The writer is obliged to disagree with this statement. While he has had cases in which adenoids were present and were removed before the papilloma was touched, it has never been his good fortune to see any diminution of the papilloma following this operation or to find any such cases reported in literature.

Baumgarten has published an interesting and instructive article which I shall quote rather freely because his conclusions coincide closely with my statements made three years ago and with my present position on the subject. He propounds an interesting theory as to the etiology of papilloma in adults which I will not go into as it is not germane to the subject of this paper. He suggests the possibility of a gonorrheal discharge from the mother at birth being a factor in etiology. As another factor he mentions bad hygienic surroundings. He also states that blonds are more susceptible than brunettes and that adenoids stand in no causative relation. There comes a time, he says, with every child who has papilloma when the power of the papilloma to recur after operation ceases. In rare cases it ceases after the first operation. In most cases, on the other hand, it loses this power only after months or years and it is immaterial whether one operates endolaryngeally or by laryngo-fissure and removes most carefully the minutest growths and destroys the base. He says further that all cases that have measles die of pneumonia. It is my own opinion that, while measles is a very grave complication, it is not necessarily fatal. Case 4, in my series, recovered from pneumonia following measles. Baumgarten keeps the tube in for six months after the larynx is free. He advises tracheotomy for children under 10 years of age even when dyspnea is slight, recognizing the disadvantages of an emergency tracheotomy. After tracheotomy one can operate unhampered by any thought of any interference with the respiration. Children who are tracheotomized bear the opera-

tion better. Hygienic surroundings, nutrition and age affect the prognosis. In children under 6 years, if symptoms of suffocation are present or come on during examination, tracheotomy is done.

A case reported by B. Robinson illustrates the evil of not leaving the tracheotomy tube in until one can be reasonably sure that there will be no recurrence. A girl of 5 years had tracheotomy on account of cessation of respiration during examination on March 19, 1904. The papilloma was removed in two operations and the tube removed a week after the second operation. The patient returned on May 14th with severe dyspnea for which she had to be intubed. The growths were removed intralaryngeally in July. On August 27th the tracheotomy wound had to be reopened and the tube inserted. In September thyrotomy was done, the growths removed with scissors and the base cauterized. The tracheotomy tube was removed in five days. In January, 1905, the papilloma had recurred and was partially removed intralaryngeally. In February laryngotomy was again done for suffocation. The tube was removed in a few weeks. The patient was admitted to the hospital in June with bronchopneumonia. She recovered from this disease but the larynx was found to again contain papillomata. These were removed intralaryngeally from time to time. An intubation tube was occasionally inserted for dyspnea. During 1906 the growths were removed nine times, the last being in November. Breathing became bad in December and she died suddenly on January 2, 1907, of suffocation before relief could be given, nearly three years after the case was first seen. One cannot help feeling that if the tracheotomy tube had been left in uninterruptedly and no attempt at removal made until the papilloma had evidently ceased to grow the event might have been a different one.

Van den Wildenberg reports two cases, 17 and 18 months old respectively, operated on through the Killian tube spatula. These cases are interesting on account of the youth of the patients, but are too recent to be of value otherwise.

Zuppinger advises intubation instead of tracheotomy when interference for dyspnea is called for. His preference does not seem well taken because of the many cases in which the papilloma grows so high in the larynx that it would obstruct an intubation tube, but no less because the presence of the tube would act as an irritant to stimulate the papillomatous growth.

Ferreri favors laryngo-fissure for papilloma even in adults. He reports cases, two adults and one child. It seems very strange that anyone can still advocate this operation. In T. J. Harris's case, reported before this association in 1905 and 1906, in which the removal of a fibroma from the larynx of a child by laryngo-fissure was followed about a year later by the growth of a papilloma, the second laryngo-fissure for the removal of this growth was more justifiable than in the ordinary case of papilloma. But even in this case I should have been inclined to proceed as in any case of papilloma.

As to the cases which C. G. Coakley reported before this association a year ago, he said himself that they were too recent to tell whether there would be a recurrence or not. Consequently it must be admitted that his results as then reported cannot be considered as proof that one would be justified in operating without previous tracheotomy in every case. I think that he would admit that, at least in cases where dyspnea was marked, preliminary tracheotomy is the safe procedure.

What has been added to our knowledge of this subject during the past three years? As to the etiology and pathology, practically nothing. Wingrave's suggestion of the presence of adenoids as a causative factor seems to lack sufficient support to be worthy of consideration. Baumgarten's, that a gonorrheal discharge from the mother at birth may be an etiologic factor, is obviously, for many reasons a difficult question to investigate satisfactorily. That bad hygienic surroundings favor this affection is certainly borne out by its comparative rarity in the children of well-to-do parents. Wingrave states that papilloma is found more frequently in the anterior half of the larynx. In nine of my cases it is stated that the papilloma sprang from somewhere in the anterior of the larynx, but was limited to that region in only five of these. In five cases the site was one or both sides of the larynx and in three others the site is not definitely stated. In one case the papillomata grew from the interarytenoid space as well as from other parts of the larynx. On the whole my experience bears out this statement of Wingrave's.

Treatment.—In treating these cases our aim is to get rid of the papilloma in the shortest time and with the least risk to the patient. Secondary desiderata are that the voice should not be impaired and that there should be (in girls at least) no

external scar. It is plain to any one who has studied this subject that no one method of treatment is applicable to every case. How, then, should one decide in a given case what method to pursue? The operation of laryngo-fissure I dismiss at once as never applicable to the treatment of this affection. The first question then which confronts one on seeing a case for the first time is shall we do a preliminary tracheotomy? Mackenzie advises tracheotomy as soon as complete aphonia is present. Garel operates without tracheotomy even when marked dyspnea exists. Others take positions between these extremes. I stated in my first paper that "these growths will not yield to any form of treatment which has been attempted, however radical, until their period of active growth has passed" or, in other words, until they have lost their power of reproduction. The age of the patient offers us little if any assistance in deciding whether the papilloma is still growing and has not lost its power of reproduction or whether it is in a quiescent state and will not recur if removed.

If, therefore, a case is seen before dyspnea has developed, a careful record should be made of the site and size of the growth and the patient should be kept under observation for a few weeks. If in that time there is no increase, removal without preliminary tracheotomy may be attempted. The various instruments used for operating with the aid of the laryngeal mirror must now give place to straight instruments and direct vision. This is obtained, under general anesthesia, the Jackson laryngeal speculum, the Killian tube-spatula or the Kierstein autoscope. I have had less experience with the first-mentioned instrument than with the other two, but find it offers distinct advantages where it can be used, not the least of these being the better illumination. For very small children, the Killian instrument is best on account of its size. After one of these instruments is introduced the larynx is sprayed with a 1 per cent solution of cocain to quiet the laryngeal reflex. Tracheotomy instruments should be ready for emergency. Great care should be exerted in removing the papilloma not to wound the mucous membrane, as any such trauma may be the site of a new papilloma. The advisability of operating without a previous tracheotomy in cases where there is dyspnea must be largely a matter of judgment in the individual case. If only a short time has elapsed since the

first symptom and dyspena is already present, showing a rapid growth, a preliminary tracheotomy should be done. When the dyspnea is marked, or when without marked dyspnea, the growth is found to fill more than half the glottis, tracheotomy should also be done before any attempt at removal is made. After tracheotomy the papilloma should not be removed at once but the larynx should be examined from time to time to determine if the papilloma is growing or not. If, after a reasonable time, usually a number of weeks, there is no apparent increase the papilloma may be removed. If it recurs the operator should again wait until it has apparently ceased to grow before attempting a second removal. Repeated operations at short intervals are inadvisable not only because they are useless when the papilloma is still growing but also because they appear to stimulate the growth. While a cure is possible in a large number of cases by simple tracheotomy and time, as some of my cases show and as shown in a series of cases reported by G. Hunter Mackenzie, quoted in my first paper, removal of the papilloma when it has become quiescent hastens the cure and shortens the time of wearing the tube. Apart from the curative value of tracheotomy there can be no question that it makes the subsequent removal of the growth easier and safer. The risk of bronchopneumonia following, immediately or eventually, tracheotomy is a real one. But in the case of a large and rapidly growing papilloma or of a papilloma recurring rapidly after a removal without tracheotomy, this operation offers the only alternative to suffocation. Intubation I consider inadvisable in any case for reasons already stated. Proper hygiene undoubtedly lessens very much the dangers of bronchial complications. The home surroundings of the child should be very carefully considered. I feel confident that the outcome in two of my cases would have been different if the patients had had better home care. On the other hand I am inclined to believe that the presence of laryngeal papilloma alone makes the child more than ordinarily susceptible to bronchial troubles.

The total length of time which the tube has to be worn varies very much. Among my cases the shortest time was ten months, the longest time was four years and seven months. The shortest time between removing the tube and closing the tracheal fistula was two months and the longest time was one year. It is not necessary to leave the tube in long after the

papilloma has disappeared, because after the tube has been worn a few months a fistula is formed which will remain open until closed by operation, so that a tube can be readily reinserted should it become necessary on account of recurrence. No fixed rule can be laid down as to the length of time which should elapse between the disappearance of the papilloma and the closing of the tracheal fistula. I am inclined to make the interval at least six months. The voice is normal in all my cured cases.

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XXXIII.

THE DIAGNOSIS OF THE OTITIC BRAIN ABSCESS.*

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Translated with special permission of the author,

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The relatively satisfactory and encouraging therapeutic results which have already been achieved in such a grave condition as brain abscess, have prompted surgeons, but more especially aurists, of every civilized land to be ever on the outlook for any new or improved methods which could contribute to an early diagnosis or a better operative technic.

Notwithstanding all previous endeavors, it often happens that the abscess is first discovered at the autopsy. Every practical surgeon can testify to the chagrin which one feels and the unjust reproaches which one heaps on himself under such circumstances, and on the other hand the real satisfaction one feels if he has been successful in recognizing and operating on a brain abscess in time. He thereby saves the life of the patient, who was unquestionably condemned to death, and his mental and spiritual welfare are advanced as well.

It cannot be denied that a marked advancement in this field of research can be shown, and that, through a proper appreciation of the symptoms and a careful analysis of the same, better results than formerly are now obtained.

The causes of a false or delayed diagnosis of a brain abscess of otitic origin are various. Every intracranial complication, which has its origin in a purulent otitis, may make its appearance in a sharply defined and easily diagnosed picture. For certainty of diagnosis however, a fully developed symptom complex is necessary. Let the contours of the picture be some-

*Schwartze's Jubilee Number, Arch. für Ohrenheilkunde, Band 73.

what indefinite or in places effaced, then the diagnosis amounts to probability only.

Many symptoms which one attributes to a brain abscess belong perhaps to a complicating meningitis. When we reflect how intimately in the course of their development, the complications are intertwined, and how the symptoms of ear diseases, functional nerve disturbances and other brain diseases can closely simulate this condition, we are prepared to extract a diagnosis from inexplicable inconsistencies.

So great a master as Schwartze was uncertain of a diagnosis which seemed to lie between an abscess of the temporal region and a meningitis. The autopsy proved it to be a cerebellar abscess. Kuhn mentions a case where all the symptoms and especially the history of aphonia, pointed to a temporal lobe abscess of the left side. Instead, the operation and subsequent autopsy laid bare a diffuse meningitis. Panse observed a somewhat similar case. Not infrequently the symptoms speak for an abscess in the cerebrum, the autopsy proving it to be in the cerebellum.

The great variations and the commonly latent course of a brain abscess make the diagnosis often very difficult or even impossible. The greatest difficulty is experienced in those cases in which the latent period is complicated, the initial symptoms very slight, and the end stage comes on very quickly and follows a rapid course.

One often meets with patients, who because of an exacerbation of an acute or chronic ear inflammation, have certain symptoms which seemingly have no connection with brain involvement. The original disease disappears entirely, or is so much better that the patient thinks himself well and returns to his ordinary occupation. He is affected now and then with seemingly unimportant symptoms, perhaps a slight headache, upon which he lays no great weight and seeks no professional advice. If, however, he does consult a physician, the symptoms are generally made light of, or not correctly interpreted.

As an example of such a course, two cases are reported. A soldier who had passed through an attack of middle ear disease was discharged from the hospital as cured. Two weeks later he suddenly died. The autopsy revealed an abscess of the temporal lobe the size of a hen's egg. In the second case, a soldier who after 33 days' treatment was about to be discharged cured, suddenly developed symptoms of acute men-

ingitis and died in a few hours. The autopsy showed a large left sided cerebellar abscess which had emptied into the cranial cavity.

In 10 per cent of cerebellar abscesses, Okada found that the patient died before any indication of intracranial complication had been found. In 14 per cent the symptoms were hidden by complications of an otitic origin, so that often only the latter were diagnosticated. In about 42 per cent the diagnosis was impossible on account of other intracranial complications, such as sinus phlebitis, leptomeningitis, and cerebral abscess which directly or indirectly were associated with the abscess in the cerebellum. Small abscesses in the cerebral hemispheres can cause marked symptoms only when they are in immediate proximity to the motor area, or near enough to have an indirect influence on it or upon some of the basal ganglia.

Not only may small abscesses remain latent, but also, as is well known, an abscess of the frontal lobe may reach an enormous size and be quiescent for a long time before any symptoms appear which give rise to a suspicion of its presence. Even in the temporal lobe, the presence of an abscess makes itself known only when its volume has so enlarged that a mechanical disturbance in the skull cavity takes place. The expanding mass causes a retarding movement on the blood stream within the cranial cavity, which is, as a general rule, characterized by pressure symptoms.

In three-quarters of the cases, fortunately, we can make an early and correct diagnosis, if we associate the brain symptoms, which are in themselves not characteristic but appear in other brain affections as well, with some pyogenic process which has preceded. Thus we must not overlook an existing or a preceding ear discharge which may be the cause of the brain abscess.

In every case therefore, where brain symptoms appear or a suspicion of the same is at hand, the present or past diseased condition of the organs of hearing must be thoroughly investigated.

Facts which should be considered as important circumstantial evidence may be stated as follows:

1. Diseases of the central cranial fossa usually lead to an abscess in the temporal lobe, seldom to abscess in other parts of the brain.

2. Diseases in the territory of the posterior cranial fossa lead usually to an abscess in the cerebellum.

3. Cranial bone disease may often be the causal factor of a brain abscess; diseases of the tympanic mucous membrane, rarely.

4. Men are more commonly affected than women in the proportion of 3:1 (385 males; 140 females).

5. The second and third decennium of life are most favorable for abscess. Their occurrence is rare before the fifteenth and after the sixtieth years.

6. In women past forty a brain abscess has seldom been found.

A very important consideration which contributes to the accuracy of the diagnosis, is the length of time that the patient has been under observation by the physician. The amount of the surgeon's experience is here, as in other realms of medicine, not to be under-valued.

In order to make certain the diagnosis of a brain abscess of otitic origin, it is necessary to recognize both its position in the cranial cavity, and the presence or absence of complications. Unfortunately it is almost impossible in all cases to give a typical picture for the course and symptoms of the disease. Some cases from the beginning take a stormy course with manifold symptoms of irritation and pressure, and lead quickly to death. Others take a very long and tedious course, and exhibit almost none or very few symptoms of value. Still others have a regular course and pass through all the phases. Cases in the last category are certainly very much less often observed than the atypical ones, and this remark applies especially to abscesses in the cerebellum. On this very account, the atypical cases play a very important role and their diagnosis now and then presents insurmountable difficulties.

In the course of a brain abscess two things are characteristic:

1. The symptoms appear scarcely ever alone.

2. They often present the character of an attack, the length of which varies greatly.

The remission of symptoms often covers such a long period of time, that it may be thought that the patient has recovered. In exceptional cases this intermission may cover a period of thirty years. Although in many cases they can remain about the same, the rule is, that each attack exceeds in severity that

which has preceded it. The intensity as well as the number of the attacks may vary. Often the first will be fatal and then again it will be the third or fourth. Between the attacks the patient is only exceptionally free from symptoms, they gradually increase in number and severity until death takes place.

The attack, like the character of the symptoms, seems to depend on a sudden increase in the intracranial pressure, due to the expansion of the abscess and to the easily compensated circulation in its neighborhood.

The symptoms of an otitic brain abscess are, as is well known, usually classified as general and local. Von Bergmann divides them into three groups according to their cause and development.

(a) Those depending on or belonging to a pus forming process.

(b) Those depending on an increase in pressure or a disturbance of functions through displacement.

(c) Symptoms of central origin which indicate the position of the abscess.

In order to make clearer the picture of the development of a brain abscess we divide the course of the symptoms into three stages:

The *first stage* or period of the initial symptoms.

The *second stage* or the latent or manifest period, which is the period of the completed abscess and the symptoms depending on it.

The *third stage* or terminal period.

These three stages go hand in hand with the pathologic processes. The initial symptoms are the result of inflammation and vary as to their intensity. In many cases they are so poorly marked that they may be easily overlooked while in others they are relatively severe. The character of the symptoms is determined by the nature and position of the pathologic processes. Local manifestations depending upon the seat of the affection are more commonly wanting than is the case with brain tumors. This fact has a two-fold explanation: In the first place the abscess is situated in a part of the brain, the temporal or frontal lobe, where local processes, of whatever nature they may be, cause but little local effect. In the second place the slow development of an abscess causes less disturbance than the pressure of a tumor.

A positive diagnosis of a brain abscess in the inflammatory

period or in other words during the initial stage or period of development is much to be desired but almost always impossible. This is explained by the fact that the symptoms during this stage are generally overshadowed by the symptoms of the accompanying ear disease. A failure to make a diagnosis at this time is without danger to the patient. The full development of the abscess is not completed, the operation would probably be without results, and would deter the surgeon from again submitting the patient to an additional procedure. It is hard to conceive of either a surgeon or an aurist who would seek for a brain abscess in the course of a stormily running middle ear inflammation.

When one considers that Schwartze found only eight brain abscesses in 8,425 cases of middle and internal ear disease, and Jansen only seven in 5,000 cases of inflammatory middle ear diseases, and that this is the experience of all who have interested themselves in the matter, we can see why there should not be a suspicion of a brain abscess during the course of an acute or chronic middle ear attack. If following a poorly marked initial stage, a shorter or longer latent period without symptom follows, it should not be a matter of surprise if the disease remains unrecognized or is only barely suspected.

In order to recognize any given disease one must, as a matter of course, have a certain symptom complex. The same holds true for a brain abscess. In order to recognize the condition, there must be certain symptoms, and in order to make a positive or even a probable diagnosis, general and local symptoms of a specific type must make their appearance. If, however, we wait for the fully developed picture as is given in most text books before beginning a surgical procedure, our therapeutic results may have a very sad ending.

Of course the presence of one or two symptoms should not result in a positive diagnosis, but to wait for all the general symptoms and still more for the local symptoms, is superfluous and dangerous for the patient. Looking back over the history of the case, and considering the principles already laid down, there are always a few symptoms of a more or less characteristic nature which stand out prominently.

By an analysis of the symptoms which accompany a brain abscess, we may conclude: (1) That, in contradistinction to the symptoms of brain tumors, where local manifestations are above all the rule, the local symptoms are not in themselves

characteristic but the general manifestations occupy a more important place. (2) That in a large number of cases a certain symptom complex is constantly present, while in others it only partly appears. There are also cases where symptoms are observed, which do not generally appear with brain abscess but only here and there occur in the very rare cases. We are not certain if they are really manifestations of the brain abscess or belong to some other complication in the cranial cavity. Local symptoms, which are dependent on the involvement of a certain part of the brain, the motor area, or the cranial nerves, may appear during the course of the disease, may be absent altogether, or may be deferred until just before death. Usually they belong to the later evidences of the abcess, and when present make the diagnosis and localization of the trouble easy. According to the present standpoint of the science there is no uniform symptomatology of an otitic brain abscess.

In the initial stage, in which the inflammation speaks for the dvelopment of the abscess, one observes that a patient, who has suffered from an acute or a chronic middle ear inflammation, or who as a sequel to these troubles has had a mastoid operation performed, will suddenly develop pain in the diseased ear or in the corresponding side of the head. Not infrequently no ear pain is present, or it is minimal; the patient complaining only of the pain in the head. Perhaps he has had one or more attacks of vomiting without accompanying nausea. In the beginning of the ear exacerbation, chills or perhaps only a chilly sensation will be observed. The chills vary not only as to their intensity but also as to their length. Repeated chills force one to the conclusion that a septic process, most likely a sinus phlebitis, has developed from the abscess. If the chills develop regularly with a fixed pause between, we have to do with a malarial fever or some complicating disease (Macewen). Generally speaking, repeated chills with a brain abscess are rare. In a child of five years in whom the author had opened an abscess in the temporal lobe, a remittent fever developed. There was naturally some fear that pyemia had developed, but further observations showed that the abscess was complicated with malaria.

In the initial period of an uncomplicated abscess the temperature is only slightly elevated (37.7 to 38.3° C.) and with it we usually find a delayed pulse, coated tongue, and general

weakness. All of these symptoms, not excluding dizziness, may accompany a middle ear inflammation without an intracranial complication coming into consideration.

The initial period varies from a few hours to as many days. If the abscess passes without symptoms into the latent period, the patient is usually lost to observation until later, when marked symptoms develop. Not infrequently the initial period passes directly over into the next, where symptoms of intracranial pressure and disturbance of the cranial cavity are made manifest. The majority of brain abscesses come under observation during this stage.

It is a self-evident fact, that to make a certain diagnosis of brain abscess, an exact knowledge is absolutely necessary of all of its symptoms as well as the symptoms of those diseases which complicate it, or can be mistaken for it. A brief recapitulation of the symptomatology of the otitic brain abscess will therefore not be out of place.

The patient during the second or manifest stage, gives one the impression of a very sick person. This impression is a peculiar one The color of the skin is pale yellow and earth like. The patient is apathetic, sleepy, gazes dreamily into emptiness and an inability for mental concentration is a regular accompaniment of the sluggishness of the cerebral functions. He answers ordinary questions unwillingly, and if a long question is framed he will often sleep before it is completed. He may answer the first part of the question correctly, then the words follow one another more slowly and articulation becomes indistinct. Speaking tires him greatly. The sleepiness very greatly resembles opium poisoning (Macewen). As the disease progresses the stupor increases until it finally becomes difficult to arouse the patient even for a moment.

The ability to use his strength gradually diminishes until it is finally lost. The patient prefers to lie in the horizontal position only. The taking of the side position is very seldom observed. Changes of position almost always bring on attacks of dizziness, nausea or even vomiting.

Many patients are loquacious. In rare cases one sees a mental unbalance of such a degree that the case will be admitted to an asylum. The intelligence is usually less affected than in other brain conditions; notably meningitis and softening of the brain (Lebert). Loss of consciousness in the course

of a brain abscess is not usually seen until just before death. In other cases it is intermittently somewhat clouded and, in the few days just preceding death, again becomes perfectly clear. There are cases with attacks of loss of consciousness but in which after a few hours the mind becomes perfectly clear. In abscesses of the cerebellum, the consciousness is hardly ever clouded and scarcely ever lost, and is extinguished only at the end of the disease. The clouding of the consciousness may increase to stupor or even coma. Delirium and melancholia with a tendency to suicide may be present.

The first and almost never-failing symptom of intracranial pressure is headache. In rare cases it may be absent. Generally it is the first and most important and constant symptom which accompanies a developing or developed abscess. The headache may be constant, remittent, or intermittent in character. As a rule it indicates the seat of the abscess but this is subject to many exceptions. Cases are known where both surgeons and aurists depending on this symptom have erred in the location of the abscess, not fiding it during the operation but at the autopsy. It thus happens that the abscess may be found in the cerebellum, while the headache is localized in the temporal region. The contrary may be also true. Exceptionally frontal headache is seen with abscess of the cerebellum. Körner cites five cases of abscess in the temporal lobe, in which the pain was usually referred to the back of the head.

The headache is of varying intensity from a slight heaviness to a pain which is almost unbearable. It may be limited to a circumscribed region or involve the whole head. Everything which increases pressure within the cranial cavity, such as alcoholic drinks, overheating, straining at stool, mental work, etc., brings forth or intensifies the headache. Children usually complain of a diffuse headache. Percussion of the head usually elicits pain at the seat of the abscess. Macewen has noted that the nature of the contents of the cranial cavity has an influence on the character of the percussion note, and Knapp heard a stronger tone on the diseased than on the well side. In the case of a cerebellar abscess the headache is generally located in the back of the head. This is an important symptom in differential diagnosis and will be observed in over half of the cases. Often, however, the pain is not fixed. Okada noted pain of a fixed type in 54 per cent of cerebellar abscesses. Politzer regards headache in the back of the head

as the one important symptom of a latent cerebellar abscess.

The headache exercises, very often, a characteristic influence on the movements and position of holding the head. (R. Müller.) The movements of the patient are careful and strained, in part as the result of cerebellar ataxia, in part to avoid shaking the head. Naturally this symptom is only of value so long as the patient is able to be about. The head will mostly be held stiffly and without movement. When the pain is in the back of the head, the position is backward and toward the affected side. The neck and back will also be held somewhat toward this side and backward. Providing a chronic inflammation of the ear is known to be present, such a position of the patient will enable the physician to make a diagnosis of a cerebellar abscess, even if he only sees the patient across the room. Hyperesthesia of the head has been seen only three times.

A rigidity of the sternocleidomastoid muscle, with pressure pain in the deep tissues and along the course of the jugularis, will be found in abscess of the temporal lobe or of the cerebellum, complicated with a thrombus of the sinus transversus.

The lowering of the pulse rate, during this period, is a symptom of great weight and immeasurable importance. It may fall as low as 30 to the minute, but usually the frequency is from 48 to 60. The pulse does not correspond to the temperature. A slowing will be observed in other brain affections, notably tumors, and in encephalitis, as a result of the elevation of intracranial pressure. There comes a period during the course of a brain abscess which is of great diagnostic importance. If the temperature is high and the pulse is slow, one can with certainty make the diagnosis of an intracranial trouble and at the same time exclude a bodily sickness as the cause. If at this time an ear discharge is present which has brought on the intracranial disease, then the temperature indicates an encephalitis or meningitis complicating a brain abscess. A meningitis caused by an infection or an infectious sinus thrombosis, gives usually a small and quick pulse. In such a case the character of the pulse depends not on the brain abscess but on the infection.

The lowering of the pulse rate is not always in proportion to the size of the abscess; small abscesses, as well as the larger ones, have not infrequently a distinct slowing action on the pulse. Perhaps the increased intracranial pressure is the principal cause of the slowing of the pulse rate, for after the

opening of a large abscess, the rate will be surprisingly increased.

In many cases of brain abscess only a slight increase in the intracranial pressure is noted, which is explained by the fact that the collection of pus takes but little more room than the destroyed brain substance, and naturally there will be only a slight slowing of the rate. Von Bergmann found that the lowering of the rate is more frequent in cerebellar than in cerebral abscess, but the author can find no difference in this regard. The difficulty of diagnosis is made greater if there happens to be a preexisting brain affection in the neighborhood (Schwartze). On the other hand, the simple lowering of the pulse rate may lead one into the error of believing an abscess is present (Koch). The author had lately the opportunity of observing a very much run down child of 9 years, who had a chronic middle ear discharge and complained from time to time of a right sided headache. The pulse was 42 to the minute. The lowering of the pulse rate was entirely due to an accompanying heart affection. Irregularity of the pulse rate is sometimes observed in cerebellar abscess.

During the latent period symptoms of dizziness are almost never absent, and are usually more marked in cerebellar than in cerebral abscesses. The dizziness is of various kinds and various degrees of intensity; changes in the position of the head or increase in the headache cause it to appear when it is otherwise latent.

Vomiting, during this period, is an almost ever present symptom. This applies to other intracranial diseases as well. In the case of a brain abscess, the symptom appears as soon as the bed-ridden patient gets up or attempts to walk about.

In the case of cerebellar abscess, vomiting continues longer and is much more common. It belongs to the most important symptoms of this disease, and has been observed in over 75 per cent of the cases. Koch mentions one case where vomiting was mistaken for a sign of pregnancy. The autopsy revealed the error and showed that the patient was not pregnant. Large abscesses, with difficulty in swallowing, indicate a pressure on the pons.

A common symptom of brain abscess is a disagreeable odor from the mouth. The tongue is usually thickly coated, often dry, brown or cyanotic, and trembles when extended. The teeth are usually incrusted. The odor from the mouth resembles very closely the odor from the ear discharge.

We have, however, the same fetor in a neglected middle ear process without an intracranial complication being present. In the case of an infected sinus thrombus the odor is, if anything, more marked than is the case with a brain abscess.

As a rule we find marked constipation, which is also observed in tumors and meningitis. In sinus thrombosis, diarrhea is the rule. There is loss of appetite, but less often an excessive desire for food. The patients have often such a strong objection to taking nourishment that a resort to artificial alimentation is necessary.

A high degree of emaciation, as a general rule, is commonly observed in the latter part of this period. This condition, together with constipation, headache and slowing of the pulse makes up a symptom complex that is of great diagnostic value. Emaciation from high fever, chills, sweating or diarrhea, speak for a general infection and not a brain abscess.

The breathing is slowed, but usually regular. In cerebellar abscess the breathing is far more slowed than is the case with abscesses of the cerebrum. Exceptionally, Cheyne-Stoke's breathing or a decidedly irregular type is observed. If there is a pressure on the medulla, there may be a cessation of the respiratory function, long before the heart ceases to beat. In meningitis and infected sinus thrombosis, as a rule, the respiration is accelerated, and only when the posterior cranial cavity is encroached upon by the meningitis, does the respiration take on the type of a brain abscess.

Retention of urine will occasionally be observed. Commonly there is found a small quantity of albumin, which disappears when the abscess is opened. Polyuria and glycosuria has been observed in a small number of cases.

Chills during this period are very seldom noted. They usually occur when a new abscess forms on the periphery of the old one.

The temperature continues, throughout the course of the latent period, very nearly normal. For a brain abscess this is absolutely typical, and is in direct contradiction with leptomeningitis and all other brain complications having their origin in the ear. In many cases the writer has noted a temporary and inconsiderable increase. A marked and permanent increase speaks always for an intercurrent complication. Oppenheim makes the statement, that where one observes a marked and persistent increase in the temperature, the probability is that there is no abscess present, or at least the abscess

is a complicated one. In the initial and final stages of the abscess one does, however, note a slight increase but of short duration only. Koch shares in this opinion as regards cerebellar abscess. Okada observed in half the cases of cerebellar abscesses a marked increase in the temperature throughout the entire course. This is only natural when one considers that a cerebellar abscess is very seldom uncomplicated.

Optic neuritis, which is always a striking symptom where increased intracranial pressure is present, takes an important place in the symptomology of brain abscess. It belongs to the more common of the manifestations of a large abscess toward the end of the second period, and the author has noted it even as early as the beginning of this period. As a rule the neuritis is not far reaching and is seldom so clearly marked as is the case with brain tumors. An atrophy of the nerve seldom follows the neuritis. The author noted this sequence in a syphilitic where every symptom pointed to an abscess, but neither by operation nor autopsy was the diagnosis confirmed.

Usually the neuritis is more marked in one eye than the other. The side of the brain affected is not always indicated by its development, for crossed neuritis has been observed. When the development of the abscess is very rapid there is not time enough for the development of the neuritis; this is also the case in cerebellar abscesses, where the surrounding inflammation is not great. (Macewen.) The writer has noted an exception to this rule. In one case, the neuritis appeared eight days after the beginning of the symptoms which pointed to an abscess. The abscess in this case was acute and passed directly over into the manifest stage. Hinsberg notes neuritis optica much more commonly in cerebellar abscesses (66 2-3 per cent). In other intracranial processes, meningitis, encephalitis, tumors, etc., the same condition is observed. It has also been noted in an ordinary uncomplicated middle ear discharge. In these cases it follows, through the involvement of the plexus caroticus of the sympathetic, which in turn leads to a vasomotor disturbance of the optic nerve and retina. The last point has not been proved. Macewen believes the cause to be a low grade of meningitis depending on the ear discharge.

Amaurosis has been observed in a single case of cerebellar abscess.

Changes in the motility of the pupils are noted following the increased pressure. In the case of temporal or frontal

lobe abscess a sluggishness of action may be recognized on the diseased side. A sluggish pupil, myosis, or mydriasis occurring on the same side as the ear discharge gives additional evidence that a collection of pus is to be sought for on the corresponding side. Sometimes one pupil is enlarged and then again it is the other. The pupil symptoms are very inconstant and a normal or contracted pupil does not exclude a cerebellar abscess. Generally speaking an enlarged pupil on one side or both sides indicates some space-occupying process in the interior of the cranial cavity. In diffuse meningitis, the pupils are at first equally contracted until the pressure symptoms appear, when mydriasis with sluggish pupils is the rule. An infected brain thrombus, with the exception of the thrombus of the sinus cavernosus, is seldom followed by disturbances of the pupils. Strabismus as well as a fixed position of the bulb is seldom seen. Ptosis more often occurs with a temporal lobe abscess than it does with a cerebellar abscess. Exopthalmus is rare and usually occurs with cerebellar abscess complicated with a sinus thrombosis.

Convulsions during the latent period are uncommon and are not characteristic, and when seen are usually found in children. The convulsions depend upon a secondary irritation of the motor tract. Severe chills are often mistaken by the laity for convulsions (Macewen).

Local symptoms may entirely fail, be poorly marked or make their appearance late. They depend on the size and location of the abscess, and have a varying value in the diagnosis. They mean much if the abscess is in the region of the motor area and very little if it is located in the frontal occipital or temporal lobe. They depend upon the destruction of the brain substance or its softening, or on the edema which precedes its enlargement. This edema may recede, and with it a disappearance of one or more of the local symptoms. The pus, in the peripheral positions, presses the nerve paths apart without causing a solution of their continuity. If the gray substance remains intact, the abscess may reach a wide limit, stretching over almost the whole hemisphere without the appearance of a single local symptom. Just in proportion, however, as it approaches the cortex or inner capsule, the local symptoms make their appearance. As an abscess can occur in any part of the brain, the local symptoms must agree with those general conditions which are characteristic for the different brain regions.

The appearance of deafness of a central origin, which can be total, in the opposite but previously healthy ear, is a very important local symptom. Usually it is impossible in the apathetic or somnolent patient to test the hearing of the sound ear, for it goes without saying that in order to determine this point we must be in position to test the condition of the integrity of the peripheral organs of hearing.

Disturbances of speech, for example, agraphia, anarythmia, optical aphasia, picture aphasia, and motor aphasia are common local symptoms. These different forms of speech disturbances can only take place when certain limited parts of the left hemisphere are involved. In cerebellar abscesses, the disturbances of speech are not exclusively due to involvement of the fibres of the nucleus in the bridge, the medulla or temporal lobe, but to a general disturbance of the intellect (Körner), or mechanical disturbance such as rigidity of the masseters. (Macewen.) A temporary loss of speech may take place. A certain psychical disturbance may be present, making the discovery of the aphasia difficult.

Cerebellar ataxia or dizziness depends upon a destruction or irritation of the vermis, from an abscess of the hemispheres which has influenced the same or from a collection of pus over the tentorium (Nothnagel). This symptom is only of value if the sensorium remains free and if labyrinth involvement can be excluded. It is a symptom which often accompanies a cerebellar abscess, as Jansen has noted in more than 80 cases. Poulsen and Heimann have shown that this symptom may be absent even when the vermis is diseased. On the other hand, we can have ataxia in hysterical patients with accompanying ear disease (Oppenheim). If we consider that in many cases, on account of the severity of the sickness, the ataxia can not be recognized, and that disturbances of equilibrium as a result of dizziness are difficult to differentiate from ataxia, this symptom when considered in the light of the demonstrated cases can not be given great weight. It is certainly not pathognomonic for abscesses of the cerebellum as the same thing has been noted, in five cases of cerebral abscesses, in meningitis serosa and meningitis purulenta.

In addition to the positive group of symptoms, which are due to certain localized disturbances in the brain, a brain abscess gives rise to a second group, the so-called distal effects. These remote or distal effects are the result of the influence of the brain irritation on another area in the brain, in a man-

ner about to be stated. These remote effects, like the original lesion, give rise to certain definite and characteristic symptoms of the same character as has already been described. As is well known, they may arise as a result of an inflammatory edema which surrounds the abscess in a more or less broad zone or as a result of the increased tension of the cerebrospinal fluid which is in turn due to the increased pressure in the cranial cavity. This tension may be equalized either through the fluids in the ordinary way or through the semisoft mass of the brain itself. Those parts which are in the immediate vicinity of the temporal lobe will be more affected than the parts farther removed.

The distal effects of a temporal lobe abscess do not extend beyond the tentorium. The effects are also limited in other directions except toward the capsula interna. If the capsule is involved, crossed pareses of the extremities, more seldom crossed paralyses and convulsions, and tonic cramps of the crossed side occur.

The hemiplegia in many cases is manifestly the result of the heightened pressure, and there is a return of the normal functions as soon as the pus cavity is drained. In other cases the manifestations are explained by inflammatory processes.

It is very important to make certain in just what order the various parts of the affected side were involved, so that one may make out whether the motor center in the cortex or the conducting path in the capsule is part of the pathologic process. If the former, the face, then the arm, and lastly the leg is affected, without a disturbance of the sensory functions, the probability is that a temporal lobe abscess has exercised its distal effects on the gyrus centralis, broadening out from below upward. The pareses are usually on the crossed upper extremity and more marked than when the legs are affected. Occasionally, a crossed hemianesthesia and a homonymus bilateral hemiopia will be observed.

Anosmia of the same side is of very rare occurrence. With it a paresis in the territory of the crossed facialis may occur, but a spasm of this nerve is seldom seen. The paresis is the result of the distal effects of a temporal lobe abscess on the inner capsule, a cerebellar abscess affecting the bridge, or the distal effects of an abscess of the bridge itself. If the paralysis of the facialis occurs as a result of a destructive process in the middle ear or mastoid process, or follows from the pressure of a cerebellar abscess upon the entrance point in the

porus acusticus internus, then the effect is noted on the same side.

It is necessary to differentiate between a paralysis of the facial muscles depending on a lesion of the cortex and an inactivity of the facial muscles depending on a paralysis of the facial nerve. In a cortical lesion an advanced paralysis is seldom observed. The patient can close the eye and retains to a certain extent the ability to show expression. The sensation of taste remains in the anterior end of the tongue. (Macewen.)

The distal effects of the cerebellar abscess usually involve the medulla oblongata and cause death through paralysis of respiration. If the abscess is in the cura cerebelli or the bridge, it can in addition to respiratory paralysis cause a paralysis of the crossed extremities, crossed facial paralysis, paralysis of both legs, homonymous hemiplegia, trismus, and weakness or paresis of the arm or both legs on the same side. One will also observe in an abscess of the bridge, copora striatum or optic thalamus, anesthesia and loss of muscular power in the extremities of the other side, perhaps due to an involvement of the posterior portion of the inner capsule A small abscess in this region may run its course without symptoms until a rupture takes place (Gowers). An alternating hemiplegia has been noted in abscess of the bridge.

Stiffness of the neck, with retraction of the head and a slight degree of opisthotonus, has been noted in an uncomplicated cerebral and cerebellar abscess (Moss, Hinsberg). More often such symptoms will be observed in meningitis, extradural abscess of the posterior cranial fossa, and in a purulent inflammation of the pons or a ruptured abscess affecting the medulla.

The deep and superficial reflexes are so irregularly affected, that no positive statements as to their value can be made. The patellar reflex varies; at times it is increased, at times diminished. and at times normal on both sides. In many cases it is absent on the affected side. Koch consideres the failure of the patellar reflex on the affected side, as an important differential point in favor of a cerebellar abscess as against a temporal lobe abscess.

Fibrillary twitchings of the muscles, although not at all pathognomonic for a brain abscess, may appear toward the last end of the latent period and in the final stages may be well marked.

A partial, but seldom total, paralysis of the oculomotorius of the affected side may be brought about when the temporal lobe abscess is of sufficient size. Usually mydriasis and ptosis with, at times, a paralysis of the rectus superior and internus, is noted. When a total paralysis of the oculomotor nerve is present we have strabismus, fixed pupil, and paralysis of all of the external eye muscles with the exception of the superior oblique and the rectus externus. The movements of the ball are, therefore, impossible with the exception of a slight excursion away from the median line and downward. As has already been mentioned, the oculomotor can be involved in a sympathetic inflammation with a meningitis.

Macewen states that when the following symptom complex is noted we can say with great probability that the abscess is located in the temporal lobe: 1. When the oculomotor paralysis is on the same side as the lesion; 2. When a crossed hemiplegia is present which starts in the face and whose characteristics indicate a cortical origin; that is when the paralysis is noted most plainly in the face, the arm being but slightly and the leg scarcely or not at all involved; 3. When a disturbance or loss of the sensations is at the same time absent.

Abducens paralysis of the same side will be noted in cerebral and cerebellar abscesses; in the latter case also a crossed paralysis of this nerve.

Trigeminal neuralgia and paralysis of the hypoglossus are rare conditions with temporal lobe abscess. Generally speaking, such affections of the cranial nerves, develop as the result of bone disease or meningitis rather than from a brain abscess.

The following symptoms have been mentioned in the literature, but their occurrence is seldom: Sensitiveness to light, conjugate deviation, nystagmus, singultus, retention or incontinence of urine, crossed pain and neuralgia of the ischias.

The organ of hearing exhibits during the course of an abscess, symptoms of an acute or chronic ear discharge, cholesteatoma, granulations, symptoms of pus retention, inflammatory softening of the mastoid bone, etc.

There has been noted, in very exceptional cases, a spontaneous emptying of the abscess, by means of a fistulous opening to the external canal or directly through the cranial bones. One almost always sees at least a trace of the preceding ear disease, but almost never a perfectly normal drum. In the vast majority of the cases, the cerebellar abscesses are found

in combination with a purulent inflammation of the labyrinth. Corresponding to the changes in the labyrinth, the cerebellar disturbances of function are of a labyrinthine nature. The tests for hearing can not always be relied upon to give us an exact differential diagnosis, for functional disturbances are not infrequently found with a cerebral abscess, and then again they may be entirely wanting in the cerebellar type. In doubtful cases, however, these tests may give us a very important starting point for our diagnosis.

When but the one ear is involved, there may appear, in rare cases, a sudden deafness in the opposite or sound ear, or a sudden improvement on the affected side. (Schwartze, Lucae, Herpin.) Körner was the first to explain the phenomena by the fact that the hearing center for the opposite ear, was situated in the temporal lobe. With a one-sided ear discharge, the sudden appearance in the previously sound ear of deafness of central origin, is a decisive symptom in the diagnosis. As this phenomenon has also been observed in the case of cerebellar abscess, Schwartze's observation, that it is the result of marked hydrocephalus internus or a hyperemia of the labyrinth, is the more nearly correct explanation.

It has been sought to prove that a shortened bone conduction has some value in the diagnosis of a cerebellar abscess, but Körner has convinced himself of the untrustworthiness of this symptom, as he has often observed it in the case of temporal lobe abscess and often found it wanting in the cerebellar type.

After the termination of the latent period, which occupies a varying length of time, the so-called terminal stage makes its appearance. This period, either through the rapid increase in the size of the abscess, through its deleterious influence on the vital parts of the brain, or through its emptying into the lateral ventricles or upon the brain surface, leads quickly to death with severe symptoms.

The patient usually dies of a severe and fatal meningitis, or the picture just before the end is that of an apoplectic stroke. Stupor and coma may come on gradually or death may be sudden. At times a leptomeningitis may develop without the discharge of pus, in which case the inflammatory zone has reached the pia. If the course up to this time has been entirely latent, or even if some of the symptoms already spoken of have been present, this has no influence on the picture of the end stage. The patient will suddenly complain

of chills and fever, the headache will increase to a great intensity, there will be one or more vomiting attacks, and then collapse, loss of consciousness, small slow pulse, Cheyne-Stokes' breathing, or perhaps complete paralysis of respiration, enlargement and immobility of the pupils, coma, stupor, and death in a few hours. In isolated cases an intercurrent disease may be the cause of death. Abscess of the temporal lobe more commonly than all others, breaks through into the lateral ventricles, and therefore the terminal stage follows the latent without an interval. This is the reason why a certain portion of these cases are only diagnosticated at the autopsy, or it is only just before death that the symptoms are recognized.

The terminal stage lasts usually about twenty-four hours, seldom longer.

Lumbar puncture as an aid in the diagnosis of the otitic brain abscess is next to be considered. This procedure is dangerous, and because of the sudden change it brings about in the intracranial pressure and the change in the amount of fluid in the cranial cavity, it may induce a sudden rupture of the abscess, and be the cause of an instantaneous death. This has often been seen in the case of brain tumors, where death occurred suddenly through respiratory failure, or paralysis. The lumbar puncture may be the cause of the breaking of the newly formed adhesions with the pia, and allow the enclosed pus to escape on the surface.

If therefore we keep these facts in mind, and also the fact that by the history of the case, we can usually arrive at a definite conclusion, this procedure must be limited to a very few cases, principally in those where the symptoms are not well marked and where it is important to differentiate between an abscess and a meningitis purulenta. Here the procedure is of undoubted value.

So far as our knowledge goes the results of the lumbar puncture have given us the following data:

1. Cloudy fluid, with pus cells or bacteria, awakens the suspicion of brain abscess.

2. A very heavily clouded fluid with many pus cells and bacteria suggests a purulent meningitis.

3. A very slightly clouded fluid with bacteria, points to a disease of the pia, but one is not certain whether we have to deal with a diffuse or circumscribed process of the meninges. A fluid of this character was observed in a cerebral abscess

and led to a very sad result, as the abscess remained unopened. (Brieger, Ruprecht, Wolf.)

4. If the fluid is free from pus cells and bacteria, then no purulent meningitis is present or we have to do with a very circumscribed process.

5. Meningitis serosa or an encapsulated purulent meningitis is to be thought of when the fluid is increased in quantity or stands under pressure.

6. An opalescent fluid will be probably due to a meningitis tuberculosa even if the tubercle bacilli cannot be found.

Although we seem to have so many certain starting points for our diagnosis of temporal lobe abscess, we are not always protected from making a mistake. The most characteristic symptoms may lead us into error. Circumscribed headache, pain on percussion in the temporal region, or the posterior part of the head, neuritis optica, dizziness, vomiting, and the general condition of the patient indicated in many cases an abscess where the autopsy proved a purulent meningitis to be the real trouble. (Clinic at Halle.) In other cases the disturbances connected with the brain were ambiguous or the clinical picture of the abscess was clouded by intercurrent complications. Comparatively speaking, the temporal lobe abscess, by a consideration of its symptoms and history, is the most easily diagnosticated. The points of selection for the brain abscess are in the large majority of cases in the temporal lobe or cerebellum.

Abscesses of the frontal lobe, which are generally rare (3 frontal, 362 temporal), fail to give any definite symptoms. This is also the case with brain tumors in this region. The first symptoms show themselves through the distal effects after the abscess has reached a certain size: Disturbances of speech if it is left-sided, and monoplegia of the opposite facialis or the arm. Occipital lobe abscesses are also very rare, and according to von Bergmann, are usually the extension of an abscess having its origin in the temporal lobe.

In children, all intracranial processes, as well as an ordinary middle ear inflammation, usually develop with symptoms of irritation and take a more active course than is the case with adults. In children an ordinary extradural abscess may be mistaken for a brain abscess. (Körner.) Local symptoms are seldom or never well marked.

The diagnosis of the cerebellar abscess presents, generally

speaking, far more difficulties than is the case with a temporal lobe abscess. The picture is poorly marked and not clear, and the diagnosis amounts to a probability only. Often the real facts of the case are only possible, when one can study the wound conditions during the opening of the mastoid or the exposure of the sinus.

Koch divides the clinical picture of the cerebellar abscess into three types:

Those cases in which marked and manifold local symptoms allow us to make a diagnosis.

Those cases with marked brain symptoms, but with few and poorly marked local symptoms.

Those cases in which neither the general nor the local symptoms are well marked, but the diagnosis is made possible by the course of the purulent inflammation, the finding at the mastoid operation and the presence in and through the dura of a fistulous opening leading to the discovery of an abscess.

It is possible to determine beforehand, whether two abscesses are present at the same time or whether one is situated in the cerebrum and the other in the cerebellum. If the symptoms do not entirely disappear after the operation and if no purulent symptoms are present to explain the remaining symptoms, we must be suspicious of a second abscess, whose location must be determined by the symptoms formerly present.

Where symptoms of intracranial complication are present and the differential diagnosis lies between a temporal lobe and a cerebellar abscess, the following points are to be taken into consideration:

1. All general symptoms are common to both locations with the following exceptions:

(a) With temporal lobe abscess it usually locates the seat of the trouble. With cerebellar abscess, it is usually located in the back of the head and sometimes in the forehead.

(b) Disturbances of the eye muscles.

2. In the cerebellar abscess, as the greater proportion of the deaths are due to failure in locating the abscess early enough, we must not wait for the special and local symptoms of the abscess in the hemispheres.

3. Naturally a cerebellar abscess may also exhibit characteristic distal effects, which consist of a disturbance of the function of the nucleus and the stems of the last six cranial

nerves, but these phenomena are far less infrequent than is the case with tumors.

4. Disturbances of equilibrium, gait, breathing, motor disturbances of speech, stiffness of the neck, trismus, nystagmus, paralysis of the facialis of the same side, paralysis of the extremities, amaurosis without atrophy of the optic nerve, convulsions in children, are all symptoms of the cerebellar abscess.

5. In cerebellar abscesses the disturbances of speech have the bulbar character.

6. Crossed, pareses, paralysis, spasms, often convulsions, hemianesthesia, amnesic aphasia, paraphasia, optico-acustic aphasia, aphasia for words, agraphia, hemiopia, ptosis, abducens paralysis, total paralysis of the oculomotorius, usually indicate a temporal lobe abscess.

7. In two-thirds of the cases of cerebellar abscesses the local symptoms are absent or poorly marked.

8. The temporal lobe abscess as a rule causes more or less local phenomena.

9. The temporal lobe abscess is most often found connected with caries of the tegmen tympani; the cerebellar abscess with caries of the posterior wall of the petrous bone or with a purulent inflammation of the labyrinth.

Here and there cases may be noted in literature where, after a collection of pus in the brain has been diagnosed, the one type of abscess was mistaken for the other. (Drumond, Garngee, Barr, etc.) Hansberg, in the case of a temporal lobe abscess, which was located in the inferior and posterior part of the lobe, maintained the diagnosis of cerebellar abscess. The autopsy showed that it was indeed a temporal lobe abscess, but lay quite close to the cerebellum.

The difficulty is increased if we have to deal with a double-sided ear discharge, which in the case of a cerebellar abscess can present insurmountable difficulties. If disturbances of speech are present, then, ceteris paribus, there is an abscess of the left hemisphere. The location of the pain, the percussion tenderness and the paralysis on the opposite or the same side, are of great diagnostic value.

The differential diagnosis between abscesses of the cerebellum and a purulent inflammation of the labyrinth is immensely difficult. Both diseases may develop together and have similar symptoms. To these belong dizziness, nausea,

headache, vomiting, nystagmus, changes in the temperature curve, etc.

Neuritis optica is common in cerebellar abscess, but exceptional in labyrinth disease. It may, however, be wanting in the former trouble. Hemiataxia and hemi-anesthesia, are never seen in purulent labyrinth disease. Disturbances of equilibrium may be wanting in both diseases.

H. Neumann considers nystagmus a very important symptom in the differential diagnosis. In abscess of the cerebrum it is almost never present. In cerebellar abscess, horizontal nystagmus has been noted both toward the diseased and toward the well side. It increases in intensity as the disease progresses to a far greater degree than is ever noted in labyrinth disease. Neumann and Barany made the important observation, that in the beginning of the labyrinth disease the nystagmus is toward the diseased side, but that later it entirely disappears. In the beginning of a cerebellar abscess, the nystagmus is toward the well side, then it may suddenly change toward the diseased side. If one observes this latter symptom, the diagnosis of a cerebellar abscess is positive, and the labyrinthine origin of the nystagmus is excluded. Besides if a cerebellar abscess is combined with a purulent labyrinth disease, the intensity of the nystagmus quickly diminishes after the labyrinth operation, if it is of labyrinthine origin, and is not at all affected, if it is due to a cerebellar abscess. The cerebellar nystagmus has a rhythmical character and has the identical features of the labyrinth type.

The differential diagnosis of the extradural abscess is in most cases not difficult. General symptoms met with are: malaise, general outward impression of a very sick person, weariness, dizziness, vomiting, etc., which are also seen with abscess. Usually these symptoms are all absent. Changes in the pulse rate are very rare. If the rate is lowered, the size of the extradural abscess is great enough to cause pressure symptoms. Choked disc and neuritis optica has been noted only four times. (Braunstein, three times; Hölscher, once.) In acute cases a slight hyperemia of the background has been noted (Braunstein). Nystagmus, convergence, divergence of one eye, dilatation of the pupils have all been occasionally seen. In very rare cases where the extradural abscess is in the central fossa, crossed pareses may be observed, and Körner has noted sensory disturbances of speech

in children in left-sided extradural abscess. In one case melancholia and in another the "idée fixe" have been described. (Biehl, Hölscher.)

If the abscess is located in the posterior cranial fossa, a stiff neck may be noted. In the majority of cases a high fever will be wanting, and if present we usually find an accompanying mastoid inflammation or a sinus phlebitis. If any change is noted it will be a lowering of the temperature. Usually the extradural, like the brain, abscess causes no change in the temperature curve. Headache is an inconstant symptom, at times it is entirely wanting and again it takes on the character of a brain abscess headache. It may be located over the ear, in the back of the head, or in other places which do not correspond to the location of the abscess, but is seldom diffuse in character. Mental or physical exertion or percussion over the seat of the abscess causes an increase in the headache, and it will usually be found to be worse at night.

Pain in the ear is often found in connection with the headache. Also as a frequent accompaniment of extradural abscess we have periostitis, formation of a fistulous opening and subperiosteal abscess. When it is remembered how seldom general symptoms and how still more rarely local symptoms are observed with extradural abscess, a proper differential diagnosis presents no difficulties. In brain abscess one will be able to recognize a more or less well marked symptom-complex, both general and local, while with an extradural abscess such a complex belongs to the exceptions. As has already been noted, a circumscribed bone change is usually found with the extradural abscess. An unusually abundant discharge of pus from the ear, which is quantitatively too great for a middle ear secretion, indicates an extradural abscess, and its presence is still more probable, if with the cessation of the discharge, the brain symptoms appear. If both diseases are present at the same time a correct diagnosis is difficult and often impossible, and if the extradural abscess runs its course with the symptoms of a brain abscess, errors are hard to avoid. In such cases the diagnosis is generally first made on the operation table.

The deep extradural abscess has the same course as the superficial, and often develops with the same symptoms or without any at all. The diagnosis of such an abscess is a

remote possibility, and it is only after the mastoid has been opened and the cerebral and cerebellar abscesses excluded that we can arrive at a definite conclusion. A fistula into the labyrinth makes the diagnosis much easier.

An empyema of the saccus endolympathicus runs its course without symptoms and often leads to an abscess of the cerebellum.

The same conditons apply to extradural abscess and pachymeningitis interna, which also cause no typical symptoms with the exception of those cases where we have to deal with typical local symptoms due to softening of the brain cortex. In such cases a differential diagnosis between a brain abscess and a subdural or cortical abscess is impossible before the operation. Subdural abscesses can by reason of their extension and at times numerous complications, cause heterogenous and inexplicable symptoms. At times, local symptoms appear as a sensory aphasia, where the abscess is located in the left temporal lobe, so that the diagnosis lies between a meningitis and a temporal lobe abscess. In pachymeningitis and the encapsulated subdural abscess in the subarachnoid space, the spinal fluid is as a rule clear or only slightly clouded. When we find such a condition by means of a spinal punction we should think of an encapsulated collection of pus, so that a distinction between a subdural abscess and a brain abscess can not be made. If after incising the dura and the inner sinus wall no pus is discovered, the question arises whether we have to do with a brain abscess or a pachymeningitis interna without the formation of pus. (Hölcher.) The subdural abscess and the pachymeningitis may be the result of the breaking through of a brain abscess, but more often is a complication of a sinus thrombosus or an extradural abscess.

Encephalitis circumscripta and brain abscess are impossible to differentate in their earlier stages, as they both arise together, and as a matter of fact the encephalitis represents the initial stage of the abscess. In the differential diagnosis no great weight should be put on the duration of the disease, but the etiology above all must be taken into consideration.

In typical cases the differential diagnosis between brain abscess and leptomeningitis is not difficult to make. Purulent inflammation of the ear may be the etiologic basis for both diseases and both may have similar symptoms. Although as a rule each disease has a characteristic symptom complex,

for itself, there are cases where one may be mistaken for the other and the difference is not therefore absolutely clear. When the leptomeningitis has a slow course it has a certain resemblance to a brain abscess. A headache which constantly reappears belongs to this group. In a few cases it is wanting or only poorly marked. Ultimately nausea, vomiting and dizziness are noted. Sensory disturbances are almost always present; they differ only in the method and time of their appearance. Their degree varies from a slight disturbance of the sensorium to the deepest coma. Under this class belong: excitement, irritability, uneasiness, sleeplessness, delirium, apathy, stupidity, etc., total or partial paralysis of the facialis, stiff neck (which is always well marked in meningitis), paralysis, myosis, sluggishness of the pupils, dread of light, later mydriasis, paralysis of the optic nerve, conjugate deviation, nystagmus, failure or increase in the patellar reflex, constipation and strangury. Macewen found neuritis and choked disc of rare occurrence, but Gowers considers them common accompaniments of basal meningitis. As is the case in a brain abscess patients are found in whom sensation is retained to the last, and the symptoms take on the appearance of an attack.

Körner noted aphasia when an exudate took place into the fossa of Sylvius. When the course of the meningitis is poorly marked the changes in temperature may be very slight or even subnormal or normal, and the pulse may also be lessened in frequency. With such a course the diagnosis presents almost insurmountable difficulties. As we have already shown, important data may be gained from a lumbar puncture. It is characteristic in leptomeningitis, that the cranial nerves are implicated to a much greater extent and more irregularly than is the case with a brain abscess. In meningitis the prodromal symptoms are of much longer duration. If the abscess lies in the region of the bridge, the cranial nerves may be widely affected, but as is well known, abscess in this region is exceedingly rare and, our therapeutic measures are without result. A false diagnosis in such a case is of no practical significance. Fever is never wanting in the acute cases and very often present in the subacute cases, its character varies, but is not at all typical. The temperature may go as high as 41°, C., but not usually over 39° C. Körner noted that a high fever predominates in meningitis of the convexity, and this type as a

rule has a more severe course than basal meningitis. The pulse corresponds to the height of the temperature, and in the end stage is small, irregular and scarcely to be felt. In rare cases it remains strong and regular.

Hyperesthesia of the skin, clonic and tonic cramps of the facialis, and convulsions and paralysis of the opposite side have also been noted in leptomeningitis. The crossed paralyses involve equally the upper and lower extremities. In the terminal stage, we find convulsions and paralyses of the extremities, a partial or total loss of speech, and always progressive loss of consciousness, Kern's flexion contractures, boat shaped contracted abdomen, and involuntary passage of urine and feces after previous retention and constipation. The urine contains albumin, peptone and sugar. Local symptoms arising in the cranial nerves are usually characteristic of a basal meningitis, while symptoms in the extremities are the signs of a meningitis convexitatis. There is often a decided increase in the white blood cells, but this symptom has also been observed in an purulent inflammation of the mastoid, and a sinus thrombosis. Herpes of the lips and entrance to the nose often occur. The meningitis which comes on with the rupture of an abscess has an extremely rapid, stormy and apoplectic course and so conceals the symptoms of the abscess itself that the latter can be overlooked.

A sudden increase in the deafness of the sound side, is more significant of meningitis than brain abscess, although this symptom, as has already been stated, occurs in the latter.

In as much as the distal effects of the cerebellar abscess takes place along the meninges, the symptoms of both diseases, for that reason, may for a time be in doubt.

In children, a brain abscess of otitic origin and sinus phlebitis, often run their course with meningeal symptoms, and have often been mistaken for a meningitis. In other cases the abscess may occur with meningitis without exhibiting any characteristic symptoms, so that the effects of the latter alone are noted.

In many cases the existing symptoms may lead directly to a false diagnosis, in that either the presence of another complication with meningitis, or a meningitis coming in as a complication with another intracranial disease, may lead to a confusion as to the real state of affairs. Brieger, for example, observed a case of cerebellar abscess, whose symptoms re-

minded one strongly of meningitis, and the same thing has been seen with an extradural abscess.

The over-preponderance of the general symptoms in the clinical picture may give the impression of a general septic disease. We must also realize that an encapsulated collection of pus may give the appearance of a general meningitis, while on the other hand the distal effects of a meningitis may be confounded with a brain abscess.

Brain abscess and meningitis serosa are closely allied and often identical symptoms. The latter may exhibit the symptoms of a meningitis diffusa, but on the whole the prevailing symptom complex is that of pressure symptoms. A characteristic symptom as opposed to brain abscess is the appearance of neuritis optica, and an early profound loss of the sensations. Stiff neck, opisthotonos, differences in the size of the pupils, strabismus, headache, dizziness, vomiting, constipation, slowing of the pulse, temperature curve, and of the local symptoms, disturbances of speech, are common to both diseases. Disturbances of locomotion, which are so important in cerebellar abscesses, are still more marked in meningitis serosa. In the latter case disturbances of sight, such as amaurosis and amblyopia are very commonly observed. Deafness, loss of taste and smell, also double-sided pareses and paralyses of both legs, both arms, both facial nerves, both abducens nerves, are not infrequently met with in the case of meningitis serosa. At times these disturbances appear on one side only, and their subsequent involvement of the opposite side indicates a meningitis serosa as against a brain abscess. Koch has observed convulsive seizures in a third of the cases. Such attacks have been noted in a cerebellar abscess, but hydrocephalus was almost invariably an accompaniment. The principal difference lies in the course of the disease. The meningitis serosa develops suddenly with severe brain symptoms, which in a few days or even in a few hours entirely disappear without leaving any traces. The manifest and end stages of an abscess on the other hand, develop steadily onward toward the end, the symptoms lasting at the most a few weeks or only a few days, whereas the attacks of meningitis may be repeated throughout months or even years. The principal differential diagnostic point lies in the lumbar puncture. In general, the meningitis serosa is more often the com-

plication of a brain abscess than it is of the ordinary purulent ear disease.

Tuberculous Meningitis and Brain Abscess.—It is to be especially noted that meningitis tuberculosa occurs especially in children under 10 years of age, and that brain abscess at this period is relatively uncommon. The disease has also been observed in adults up to thirty years of age. The meningitis is usually accompanied by general tuberculosis, but here and there one sees cases where the original trouble is lost sight of, and the meningitis appears as an apparently primary disease. Tubercular meningitis in children is usually accompanied by preliminary symptoms. They lose flesh, are weak, depressed, irritable, the sleep is uneasy, and they often complain of headache, which is increased by bodily work or mental exertion. Such a condition may be of weeks' duration. Then apparently without any couse, vomiting appears, which is usually accredited to an indiscretion in the diet and the headache becomes more violent. Symptoms of brain involvement appear which have the same character as a brain abscess, viz: somnolence, delirium, stiff neck, differences in the pupils, dizziness, convulsions, obstipation, and lowering of the pulse rate to 40 to the minute. The temperature increases a little, to 38.5° C. Local symptoms such as aphasia and poorly marked neuritis optica have been observed. The characteristic choroidal tubercles may be absent. At the end of the first or in the second week of the disease an involvement of the cranial nerve begins, shown by strabismus, inequality of the pupils, a slight degree of ptosis, and a paresis of the facialis. The somnolence passes into a coma, the head will be drawn backward, if this is not already the case, and a rigidity of the limbs may be noted. Commonly local convulsions or paralysis, hemiplegia, or paralysis of the arm or face, seldom of leg or face, will appear. The paralysis may be lasting or temporary. The pulse becomes rapid, 140 to 180, the respiration difficult and irregular, and the patient finally dies in coma. The temperature is either not elevated or is subnormal. At times just before death an apparent improvement takes place. If only the convexity of the brain is affected, which is seldom, the symptoms of cranial nerve involvement are wanting and vomiting is rare, and delirium, convulsions, and rigidity of the extremities become the prinpipal symptoms.

On account of the many symptoms common to these two diseases, and because of the possibility of an accidental tuberculosis in other organs, the difficulties of a differential diagnosis in a patient with a brain abscess of nontubercular origin, are often insurmountable. This is especially the case when one reflects that in children an acute middle ear inflammation is often seen with symptoms of headache, vomiting, fever, delirium, dizziness, strabismus, and a more or less well marked neuritis optica; and that the brain abscess often runs the course of a meningitis. In the best of cases, the diagnosis can only be probable.

If a child with no hereditary tuberculous tendencies is seized, during the course of an acute or middle ear inflammation, with attacks of severe head and ear pain, vomiting and other symptoms of intracranial pressure, with normal or subnormal temperature, with convulsions or loss of consciousness during which the mind remains clear, the symptom complex speaks for a brain abscess. In such cases stiffness of the muscles of the neck, pupil differences, fixation of the bulb, disturbances of equilibrium, and a staggering gait, indicate cerebellar abscess. A well marked neuritis optica at the end of the first week of sickness is also in favor of a cerebellar abscess. The diagnosis of a tubercular meningitis is made positive if the tuberculin reaction in the eye is positive, and if we find tubercle bacilli, lymphocytes, and a slight clouding in the spinal fluid. The tuberculin reaction and the presence of tubercle bacilli often fail. The puncture of the arachnoid sac during the mastoid operation may also be used as a diagnostic agent.

In large children and adults, the symptoms of a tubercular meningitis are the same with the exception that the prodromes and convulsions are rarer. As with abscess, the pulse and temperature remain the same. Sudden unconsciousness, delirium, marked stiffness of the neck muscles, and crossed disturbances of motion and sensation are more in favor of a meningitis, whose character, by an investigation of other organs and a careful review of the clinical history, may be more or less accurately ascertained. If tubercular caries of the middle ear and the adjacent parts are present, the meningitis is probability of tubercular origin. An abscess may, however, be present at the same time, but with the above mentioned general and local symptoms and a sound organ of hearing

and with symptoms of tuberculosis in other parts of the body, a brain abscess is decidedly out of the question.

Virchow was the first to explain in what manner a collection of tubercles in the brain could lead to the formation of an abscess. Such a tubercle has its point of selection between the gray and the white matter. If the caseous contents begin to break down and liquefy, a thing which happens but seldom, we have in the middle of the tubercle a small space filled with a cloudy whey-like fluid. At times one notes that the tubercles are surrounded by pus or the entire mass is of a purulent nature. Such an abscess may be easily mistaken for a brain abscess of otitic origin, and is only to be differentiated by the presence of more or less tubercle bacilli. The symptoms are precisely similar to those of a brain abscess, but concomitant changes of a tubercular nature in the lungs or other bodily organs, will generally be found. The writer observed a brain abscess, with lung tuberculosis as a complication.

It would be difficult to make a mistake in the diagnosis between a brain abscess and meningitis cerebrospinalis epidemica. If we consider, in connection with the ear pain, the symptoms of the meningitis: the severe headache, retraction of the head, cutaneous hyperesthesia, delirium, pain in the back, muscular rigidity, pain in the neck, muscles, herpes labialis, even leaving out of consideration the epidemic and infectious nature of the disease, an error as to the real nature of the trouble is almost impossible.

Sinus Thrombosis and Brain Abscess.—A confounding of these two troubles is uncommon. In most cases, at least in the beginning, headache and vomiting will accompany the thrombosis. The headache is at times diffuse at times sharply circumscribed on the diseased side or referred to the ear itself. In cases where the condition is not complicated with a brain abscess or meningitis, the consciousness is not at first disturbed, but later it may be clouded to a slight degree. The opposite of these conditions has been noted in scattered cases. The author observed several cases of septic thrombus where the autopsy showed a well marked basal meningitis, in which not taking into account the stupor depending upon the infectious process itself, the consciousness was not at all disturbed during life, but only a few hours just before death. Psychical depression, a slight degree of stupor and a general loss of strength are very common. Neuritis optica is seldom ob-

served. Körner has never observed it in cases of the transverse sinus, but it does appear in diseases of the cavernous sinus, and Jansen considers it here of diagnostic value.

The most important differential symptom which separates an infected sinus thrombosis from a brain abscess, is the characteristic septic fever, with its accompanying chills, sweating and rapid rises and falls in the temperature (41.5° to 36.2°). In children, the chills and sweating may be wanting, and the fever itself, especially when metastases are present, may not show the characteristic curve. The pulse corresponds to the temperature, and when a sepsis is present, the acceleration will be noted during the afebrile intervals. A slowing of the pulse to 42 has been noted in rare cases. (Kessel.)

Metastatic brain abscesses are very rare and usually multiple. A single abscess only may be present, which is therefore operable. The metastatic abscesses originating in a purulent ear discharge, are usually peripheral, and occur especially in the joints, subcutaneous tissues and muscles, seldom in the lungs, and rarely in other organs.

External manifestations which characterize a phlebothrombosis, are involvement of the nerves which pass through the foramen jugulare (vagus, accessorius, glossopharyngeus), as well as the hypoglossus. In addition we may find a painful edema of the posterior border of the mastoid, edema of the eyelids (in diseases of the sinus cavernous), a marked filling of the veins of the forehead on the diseased side and unequal fullness of the external jugulars. Often a hard painful cord-like swelling of the thrombosed vein and the infiltrated connective tissue may be distinctly felt. Movement or turning the head toward the sound side causes pain. Hoarseness, aphonia and difficulty of breathing are often present. Localized convulsions in the region supplied by the accessorius, paralysis of the muscles involved in the act of swallowing, paralyses of the muscles of the soft palate, all come into consideration.

Plugging of the sinus cavernosus makes itself known through a passive hyperemia in the region of the ophthalmic vein and the disturbances of one or more nerves which stand in close relation to it. A high continuous fever, a weak and rapid pulse, disturbances of consciousness and a marked loss of strength make up the symptoms of a septic intoxication.

In children an uncomplicated sinus thrombus can, at times,

run its course with the so-called meningeal symptoms. In such a case a certain diagnosis is not to be made, but a lumbar puncture will help to clear the matter up.

If a sinus thrombosis is complicated with a brain abscess, or if at the same time a leptomeningitis is present, the symptoms of the general infection are so well marked that the symptoms of the complicating disease are to a certain extent clouded. In many such cases the brain abscess is small and develops after the formation of the thrombus. (Macewen.)

Brain Abscess and Apoplexy. — The end stage of the brain abscess or its final rupture into the side ventricle or upon the surface of the brain, has many points of similiarity with an attack of apoplexy. This is especially true if the etiologic cause of the brain abscess has been disregarded. On the other hand, apoplexy, when accompanied by a purulent middle ear disease, may be mistaken for an abscess. In both diseases two main sets of symptoms dominate: general and local. The attack can be accompanied by preliminary symptoms, such as headache, dizziness, slight physical disturbances, slight disturbances of speech, which with the presence of a purulent ear discharge cause one to be suspicious of a latent running or ruptured abscess. Apoplexy, however, comes in the latter periods of life, while abscess is usually seen during the first three decenniums. In apoplexy, of the general symptoms, loss of consciousness, and the local hemiplegia, are the most common symptoms. The apoplectic attack comes on suddenly, the patient collapses into a comatose condition, and for a shorter or longer time is wholly or entirely unconscious. Movements and sensations are entirely blotted out, and the patient may die in a few hours or even a few minutes. The pulse is at first slowed, small and hard to feel. At times it is accelerated. About an hour after an attack the temperature sinks as low as 30° and remains so until death or rises again to normal. A convulsion may accompany the attack, but it is as a rule wanting. In certain cases the patient may recover, the general symptoms recede and the hemiplegia alone remain. The rupture of an abscess will almost always be accompanied by a chill, increased temperature, violent headaches and vomiting. The pulse is greatly accelerated, and the other symptoms, already mentioned, may appear. The attack is invariably fatal. As a rule it can be ascertained that with the brain abscess, certain general symptoms, and

often local symptoms have been present for a longer or shorter period, while in apoplectic cases these are generally wanting or when they did appear were of short duration and not characteristic.

Brain Abscess and Tumor.—Here the etiology is of great importance. The very absence of a specific etiologic factor would speak for a tumor. Distinct local brain symptoms and a slow gradual increase in the course of the same, especially with paralysis of the cranial nerves, a high degree of neuritis optica, with often progressive blindness, at times hemianopsia, general and local convulsions, general muscular weakness, attacks of loss of consciousness, all favor a new growth. The receding of severe symptoms also indicates a tumor. The temperature never vacillates, but as we have already shown the course of the abscess can be an afebrile one or the curve may even drop below the normal. In the latter affection, however, there will be slight changes in the temperature with generally an increase toward the end, following the appearance of complications.

The rapid development of acute or severe brain symptoms after only slight signs of a brain affection have been noted, indicates an abscess. The symptoms common to both troubles are,—headache, dizziness, vomiting, stupor of the mental faculties, neuritis optica, uncommon in abscess and not so well marked as in tumor, and lastly cerebellar ataxia in the case of cerebellar tumors. Neuritis optica is sometimes a very early symptom, especially in tumors of the cerebellum and diseases of the copora quadrigemina. In tumors of the cerebrum it makes its appearance late.

Convulsions often appear in the case of a tumor before a suspicion of the growth is present. Although in the case of a tumor, the development of the symptoms is slow and gradual, the course of the individual symptoms is not always equally so. In slow growths, as is the case with an abscess, the process of the development may be intermittent or even stationary. Pressure symptoms may appear quickly in the case of a tumor; a healthy facial nerve, for example, can in a few days be completely paralyzed. In other cases a rapid increase in the symptoms occur as a result of a local meningitis caused by the tumor. Commonly there follows a temporary or lasting hemiplegia, following a convulsion, or even without one. In the cases of recognizable syphilis, it is prob-

able that the tumor is of this nature, and eventually it forms a gummi which under certain circumstances may break down and in this manner give an indistinct picture of a brain abscess. The diagnosis, as is the case in the tuberculous, may be only an indefinite one.

The intracranial aneurysm also forms a new growth and gives rise to symptoms of a tumor of an entirely different nature. The only difference is the presence within the cranium of an audible aneurysmal murmur.

In very rare cases a new growth can take place in the presence of a purulent ear disease, and this may cause the diagnosis to become a matter of great difficulty or even impossibility.

Hysteria may be mistaken for an abscess, when it occurs in connection with an ear discharge or a purulent affection of the temporal bone. This is especially the case if the patient has been under observation for a short time, and if headache and vomiting are present. The sudden outbreak, the failure of all symptoms of organic disease or neuritis optica, the method and appearance of the objective symptoms, as well as the circumstance that hysterical individuals have a tendency to imitate allows one to make the correct diagnosis. Hemianesthesia, disturbances of the special senses without a concomitant motor paralysis, are among the most infrequent of the symptoms observed in brain abscesses or tumors.

In females, and perhaps where other hysterical symptoms are present, it sometimes happens that a brain abscess is mistaken for hysteria. In order to exclude diagnostic errors as far as possible, the apparent hysterical symptoms must never be allowed to influence the diagnosis, until an organic brain affection can be positively excluded. The best protection against diagnostic mistakes is an exact knowledge of hysteria, and an exact knowledge of the brain abscess, as well as a rich experience in these fields. Upon the other hand, it must not be forgotten that the diagnosis of hysteria presents, in most cases, no especial difficulty if we take into account the manner and origin of the disease, the course of the same, and the presence of certain characteristic symptoms. Experience has taught us, however, that hysteria may give rise to diagnostic errors, and to determine whether certain symptoms belong to the hysteria or not, presents problems which are difficult even for the experienced observer to solve. If it is

necessary in a given case to make a positive diagnosis, the principal thing to do is to seek for the appearance of the stigmata of the disease. These consist of psychic and somatic disturbances, anesthesias, hysterical zones, attacks of convulsions, etc. In seeking for stigmata, organic lesions, especially those characteristic for brain abscess, must be excluded. It is to be regretted that many symptoms which are characteristic for brain abscess are often found in hysteria.,

With the exception of certain symptoms, the method of origin and the further development of the disease, allow one to come to a fairly definite conclusion as to its nature. In hysteria the course of the disease is very seldom regular. Severe disturbances may suddenly disappear without improvement of the general condition. Attacks which suddenly develop with great violence, as well as a sudden cessation of long standing symptoms, point to hysteria. A number of hysterical symptoms, such as anesthesias, hyperesthesias, paretic conditions, contractions, tremors, etc., may suddenly run from one side of the body to the other. The grouping together of certain symptoms often gives us certain diagnostic starting points, as for example: the hysterical hemianesthesias do not occur with hemiopia, but the hysterical paraplegias do occur with anesthesias which are confined to the legs.

An example from von Boissard serves to show how easily hysteria can be mistaken for a severe brain complication. A female patient, 45 years of age, was suddenly attacked with severe headache and vomiting of a cerebral character. The pupils were small, the pulse slowed to 48, and obstipation was present. The temperature was normal. As a member of the family had died of pulmonary tuberculosis, the suspicion pointed to meningitis tuberculosa. After a few days the condition improved, a right sided hemianesthesia appeared, and immediately the hysterical nature of the trouble was apparent.

The author, a few years ago observed the case of a young man of 26, upon whom a mastoid operation was made, following a right sided chronic ear discharge. The patient complained continually of symptoms that pointed to a brain abscess. At the hands of a surgeon he was trephined twice without result, and the brain substance explored. The entire condition was that of a hysterical person who rather expected further operations on the skull.

Neurasthenia, especially when an ear discharge is present,

may often give rise to the suspicion of a brain abscess. This latter disease may often closely simulate the cerebral type of neurasthenia, especially in its preliminary period. In the further course of the trouble, there comes with these doubtful symptoms, those which can only be explained as depending upon an anatomic change. Just in proportion as the symptoms depending upon an anatomic basis are not present, just in that proportion must, *ceteris paribus,* neurasthenia be taken into consideration. It is not correct to say that symptoms of neurasthenia are of a fluctuating character. In many cases they are very obstinate, especially dullness in the head, the nervous asthenopia, the decrease in the ability for mental work, weakness in the legs, etc., symptoms which can not be looked upon as harmless as they are often seen in brain abscess. On the other hand, at least a certain proportion of the brain abscess symptoms show a marked degree of fluctuation. In neurasthenics, the headache seldom reaches a great intensity without special cause, and less seldom continues such intensity over a long period of time. Percussion tenderness of the skull and dizziness are not present. Vomiting, a symptom which is always present with brain abscess, is always absent in neurasthenia. Mental depression, remarkable changes in the voice, transient changes in the strength, and difference in the size of the pupils, are common to both diseases.

Hyperesthesia of the scalp is common with neurasthenia, but rare in brain abscess. Aphasic changes in speech are wanting in neurasthenia, but in rare cases a paraphasia, word amnesia, or paragraphia may be present. Of the motor disturbances, a long continuing paralysis of the extremities fails absolutely. Paralytic disturbances of the facialis, and of the eye muscles, myosis, reflex immobility of the pupils, changing reaction to light, slight pareses in the region of the above mentioned nerves, must be considered as all speaking against neurasthenia. On the average the pulse is 80 to 100. In neurasthenia, all changes of hearing, tuning fork tests and bone conduction must be considered of labyrinthine nature, and, according to Babinsky, of central origin. They are only noted in traumatic neurasthenia.

Exceptionally, coma diabeticum may, in a person suffering from an ear discharge, bear a striking resemblance to a brain abscess in the end stage, and thus give rise to diagnostic errors. This is apt to be the case, if the attack comes on sud-

denly, and if the patient has been seen for the first time. The patient is entirely unconscious; the coma is seldom broken by periods of consciousness. Occasionally unmistakable clonic convulsions appear, and mydriasis, fixation of the pupil, half opened lids, small and quick pulse, temperature below normal, deep inspiration with short expiration, and growing cyanosis, are observed. The history usually shows previous headache, loss of sleep, uneasiness, anxiety, dizziness, and a continuous feeling of intoxication. The penetrating odor of acetone, directs us to the proper differential diagnosis. A further diagnostic point is the presence of sugar in the urine and its exceptionally strong reaction to chlorid of iron. Naturally a diabetic coma and a brain abscess may be present at the same time, but under these circumstances an inability to make a correct differential diagnosis is of little practical value.

Uremia, occurring together with a purulent otitis, may give occasion to a confusion of diagnosis. The first attacks, which make themselves known by loss of consciousness, coma, and tonic or clonic convulsions, are usually preceded by headache, dizziness, apathy, a slight dulling of the sensations, an increased desire for sleep, and not infrequently nausea, vomiting and diminished pulse rate. With an abnormally high and at other times an abnormally low temperature, 42°-30° C. paralyses, contractures, and twitching of the muscles, are but seldom observed and still more infrequently mania, melancholic depression, and delirium. A consideration of the reasons for the suppressed or decreased secretion of the urine, as well as an investigation of the fundi for retinitis albuminurica, will almost always guard against a mistake in diagnosis. The amaurosis, which not infrequently develops without any anatomic ground, demonstrable with the opthalmoscope, and which may also completely disappear in a short time, is a condition never seen in brain abscess. Here also, in connection with, but not depending on the disease which gave rise to the brain abscess, we may have a brain abscess, and the diagnosis may of course present great difficulties, and often make it almost impossible to arrive at the correct solution.

The sequence of the symptoms in typhoid fever is so peculiarly characteristic, that a mistake can scarcely be made in its recognition. However, this regularity in sequence, especially in the temperature curve, can not always be expected, and the patient must be under observation for a certain period of

time. It is easier to confound typhoid with pyemia than with brain abscess. We must never lose sight of the fact, that we may have a typhoid with a low temperature, slowed pulse and preponderating brain symptoms, and thus give occasion to an error. An exact knowledge of both diseases, a continuous and accurate observation of the course of the disease and the agglutination will be enough to protect one from all diagnostic failures.

Up to the present writing the writer has been twice consulted by internists, where the suspicion of the brain abscess was present. An exact consideration and investigation of the symptoms convinced him that he had to do with a typhoid fever. In the one case there was no ear discharge present, the physician basing his diagnosis on circumscribed headache, percussion pains, boring pains in the ear, torpid mentality, vomiting and heightened fever.

Epilepsy is not difficult to differentiate from brain abscess. The epileptic attack is characterized by sudden entire loss of consciousness, sudden falling to the ground, and a well marked facial pallor which rapidly changes to a red or cyanotic color. It is very often accompanied by a piercing cry and rigidity of the body, and tonic and clonic convulsions quickly appear. An entire loss of sensation and every psychic process is observed. The pupils are dilated, and do not react to light, trismus, opisthotonos, a strongly beating heart, and blood flecked lips, are all to be noted. After a longer or shorter period, from 10 to 15 minutes, but at most an hour, the attack ceases. Epilepsy occurs usually before the twentieth year, later it is of rare occurrence. Loss of consciousness and convulsions are also noted in abscess and the attack can also disappear but neuritis optica will be present, the convulsions will not be so general and will be accompanied by paralysis.

It is possible that a confusion could arise between brain abscess and intermittent fever. In any event an uncertainty in such cases could last for a short time only, as for instance in rare cases of malaria, which have a course without fever or enlargement of the spleen. The high temperature, the continuous chills preceded by a feeling of heat, and the examination of the blood, would all guard against a mistake in diagnosis. As is well known, chills and fever, during the manifest stage of the brain abscess are practically unknown and

can occur only in the prodromal and end periods. In the latter case, death invariably follows, while a death during an attack of chills and fever with malaria is practically unknown.

As a curiosity, the writer was twice consulted in cases of circumscribed and rheumatic pain in the region of the temporal bone, which came on in paroxysms and in the absence of any ear disease, gave rise to a suspicion of brain abscess.

From all that has been said, the diagnosis of brain abscess can be made on the average with a certain degree of certainty, but here as in all other branches of medicine, cases occur which are contradictory, not well marked, and difficult to make clear. When errors of diagnosis are made, or when the abscess is not recognized, then the cause lies in the disease itself or in the similarity of the remaining endocranial complications, which depend upon a purulent process arising in the organ of hearing. In addition many of these complications make their appearance at the same time with the abscess, or are caused by the same, and therefore cloud the picture of the abscess itself. Finally we must take into consideration the similarity of the symptoms of the brain abscess, with other general diseases where no pus process is present.

It is to be hoped, that with the progress of science, the methods of determining the brain abscess will also be perfected. It is doubtful, whether in the future, a brain abscess can always with certainty be diagnosed, for errors will always be a part of man's nature, and to that end we must strive as fas as possible to eliminate them by exact observation, rich experience and correct judgment.

ABSTRACTS FROM CURRENT OTOLOGIC, RHINOLOGIC AND LARYNGOLOGIC LITERATURE.

I—EAR.

The Treatment and Prognosis of Chronic Middle Ear Discharge.

RUPPERT, Munich (*Münch. med. Wochenschr.*, No. 21, 1908). This paper, appearing as it did just before the meeting of the German Otological Congress in June, 1908, was very favorably received there, voicing as it did the sentiments of the famous head of the Munich Clinic, Prof. Bezold, where conservative treatment of middle ear suppurations is carried to its extreme limits and operative interference is the exception. The author, one of the assistants in the klinik, recognizes the fallibility of statistics based on ambulatory clinical material, where the attendance is always irregular and where the assistants are always changing, and takes instead the statistics from Dr. Schiebe of Munich, a former pupil of Prof. Bezold. In the seventeen years covered in the investigation, from 1889 to 1906, the author acted as assistant for the last four. Bezold's boric acid powder treatment was the only method used. The patients were treated every day until the abatement of the discharge, then two to three times a week. Under the term "Chronic middle ear discharge" the author places only those cases of inflammation of the middle ear cavities where a perforation remains continuously open and discharges for years, following the original acute attack, or where the perforation has remained opened because of scar tissue and after years sets up a chronic discharge. Even cases of acute middle ear suppuration which continues a full year before resolution, were not considered in these statistics, as the perforation finally closed, when the discharged ceased. In the seventeen years 1,118 cases of chronic middle ear discharge were treated; of these 38 or 3.4 per cent showed complications from the beginning. The remaining 1,080 were uncomplicated. Of this number 592 or 55 per cent had a central perforation; the remaining were either in Schrapnell's membrane or involved the annulus tympanicus. The minute description of the technic of the treatment, which because it is a type of this method as it is so commonly carried out here in Germany, is repeated here in

detail. After a thorough syringing out of the external auditory meatus with a 4 per cent solution of boric acid and Politzerization, a fine, curved cotton applicator was used very carefully to dry out the middle ear by introducing the cotton directly through the perforation. An insufflation of the most finely powdered boric acid was then made.

In 10.1 per cent of the cases with central perforation the formation of granulations and polypi was noted. These were removed only with the snare or curette; the author claiming a certain danger of facial paresis or even total deafness through the use of the chemical cauterants.

The disagreeable odor disappeared after the first treatment or at latest after the second. Three hundred and seventy-one cases or 93.9 per cent were completely relieved for a period of always more than a half year following the cessation of the treatment. In 18.5 per cent the discharge ceased after the first treatment. In 28.1 per cent the discharge ceased inside of a week. In 28.6 per cent the discharge ceased inside of a month. In 8.6 per cent the discharge ceased inside of two months. In 3.5 per cent the discharge ceased inside of three months. In 3.5 per cent the discharge ceased inside of six months. In only 2 per cent did the treatment last a year, and in four cases or 1 per cent the treatment was carried out for a longer period of time. In 24 cases or 6.1 per cent the discharge continued in spite of treatment covering years. An interesting analysis of the cases where the healing covered a period exceeding six months is given. Some of the cases were treated at home, others lived under very unfavorable conditions and in still others a strumous diathesis seemed to play an important role in the delay.

Most of the 124 uncured cases have been under observation from 5 to 18 years and none has developed complications or shown a tendency to recurrence of the fetid secretion. One of the cases which had been so long in treatment was operated on (not by the author), but the discharge never ceased.

Of the 395 central perforations two were operated upon by the author. In one with a small round central perforation which after more than six months' treatment, discharged a strongly smelling crumbly pus, the radical operation was performed. No cholesteatoma or necrosis was found and the author believes that a little more patience would have led to a complete conservative healing.

The second case was a hysterical individual, whose alarming symptoms pointed to a meningitis serosa and involvement of the labyrinth. Nothing but a diseased condition of the mucous membrane was found at the operation.

In seventeen years 489 cases of perforations involving the annulus were observed. Of these, 359 remained under treatment. Here anatomically and pathologically a new set of conditions was to be combated. The Hartmann's tympanic tubes were used to wash out the attic and antrum; a drying with cotton on the end of a carrier with a slight bend upward, followed, and the treatment was concluded by blowing powdered boric acid through the perforation into the antrum itself.

The formation of polypi was much more common in this group, 39.8 per cent as compared with the central perforation cases. The extraction of the ossicles was not found necessary in a single instance. The author saw many cases of so-called caries, with cholesteatomous masses in the antrum go to complete healing after the conservative treatment of the discharge!

Of these 359 cases, 82 were perforations in Schrapnell's membrane. The formation of cholesteatoma was noted in 53.8 per cent of the perforations which involved the margo tympanicus and in 70.5 per cent of the cases with the perforation in Schrapnell's membrane.

As was to be expected, the time of healing in this group was notably longer than in the first: 8.9 per cent were cured by the first treatment; 18.4 per cent inside of eight days; 25.6 per cent within one month; 11.7 per cent within two months; 5.6 per cent within three months; 7.6 per cent within six months; 7.2 per cent within twelve months; 4.5 per cent more than a year; 10.6 per cent remained uncured. In the 359 cases, 357 were healed by conservative methods and 7 came to operation. The remarkable results achieved here and the general discussion of the same theme later in the German Otological Congress, gives one a good idea of the conservative methods now in vogue in Germany. *Horn.*

A Case of Mastoiditis with Brain Complications.

KNAUSE, New York (*Archives of Otology*, Vol. xxxvii, No. 2). A young man gave a history of earache lasting one week. In spite of a paracentesis mastoid tenderness persisted and edema developed over the mastoid and extended backward toward the occiput.

On operation a subperiosteal abscess was drained, a perforation was found in the region of the zygomatic cells and another over the tip. His condition was uninproved. On the third day after operation he was restless, at times delirious, temperature 103° F., pulse 104,, respiration 24. There were tenderness over and rigidity of the posterior cervical muscles. Beginning choked disc on the left side. Cerebration was slow and he had agraphic aphasia. On lumbar puncture the fluid came slowly, was slightly turbid and free from bacteria.

Seven days after the mastoid operation he had a tonic convulsion with marked retraction of the head, this was followed by a paralysis of the lower branches of the right facial nerve. Next day rectal temperature was 97.8° F., pulse 54, respiration 14, and an exploration was made for brain abscess. The incision for exposure of the mastoid was carried forward and upward for about two inches and after exposure of the dura a narrow blade was passed in various directions but without result. His general condition was much improved after this brain exploration, then he contracted erysipelas; from this he was convalescing nicely, being even able to be out of doors, when he suddenly died. On autopsy the dura was found very adherent to the left temporosphenoidal lobe. This lobe was yellowish-green in color, soft and mushy and on section about half an ounce of greenish pus escaped.

There was acute parenchymatous degeneration of the heart muscle, acute miliary tuberculosis of the lungs, acute tubercular splenitis, acute parenchymatous nephritis and fatty cirrhosis of the liver. The cerebral abscess was opposite the kidney-shaped opening made in the skull and its wall was not more than one-half inch in thickness. *Campbell.*

Report of Two Cases of Sinus Thrombosis Complicated by Cerebral Abscess in the Temporo-Sphenoidal Lobes.

DABNEY, Marietta, Ohio (*Archives of Otology*, Vol. xxxvii, No. 2). CASE 1. A man, aged 48; one month before coming under observation developed an acute suppurative otitis media of the left ear. There has been continuous pain in the ear, over the mastoid and more or less pain of the entire head. There is mental hebetude. Temperature 101° F. and the ear discharge is blood-streaked. Smears show streptococci. Drainage appears to be insufficient. There is sagging of the posterior-superior wall and swelling of the tissues over the mas-

toid. On operation pus was found in the antrum and granulation tissue filled the bony trabeculae. On clearing out the granulations in the tympanic cavity there was found an erosion in the roof of the tympanum, but the dura seemed to be intact.

On the third day after operation he felt very well after the dressings were changed. Babinski reflex slight, Kerning's sign positive. The pulse, however, was rapid. On the fifth day he could not be roused, both pupils were dilated and there was motor paralysis over the entire right side of the body. Brain abscess was diagnosed and located, probably, in the region of the lenticular nucleus, where the motor tracts cross.

Under anesthesia the perforation in the roof of the tympanum was enlarged and the dura incised outward and backward along the floor of the middle fossa. A grooved director was then passed upward and inward into the brain substance for a distance of one and one-fourth inches and liberated about two drams of fetid pus. This pus contained streptococci. Two days later the patient died without having regained consciousness. Autopsy was not permitted.

CASE 2. A boy, aged 15, had had a number of attacks of purulent otitis in the right ear. The present attack began ten days ago. He has the appearance of one suffering from septicemia. Temperature 103° F. There is edema over the mastoid and the slightest pressure there causes excruciating pain. The posterior-superior canal wall sags and the sanguinolent pus contains streptococci and Weichselbaum's diplococci.

The entire mastoid structure was found filled with the products of inflammation. The antrum was filled with cholesteatoma and granulation tissue protruded from an erosion in the roof of the antrum. While curetting this fistulous opening a small subdural abscess was opened and drained. The dural opening was enlarged and an area of necrotic brain tissue about the size of a large hazelnut was found and removed.

During the next 24 hours the patient had two chills and sweated profusedly. The temperature ranged from 100° F. to 105° F. and pulse 50 to 106. Along the course of the internal jugular vein the neck became very tender and edematous, and he developed a cough with prune-juice expectoration.

Sigmoid sinus thrombosis was suspected. The internal jugular vein from the clavicle to the skull was resected. The sinus was uncovered and its parietal wall was found thick-

ened and covered with a layer of plastic lymph. As the sinus was about to be opened the patient failed so rapidly that operation was discontinued.

For the next two days he was rational at times, then this would be followed by maniacal delirium. The pupils were dilated There was convergent strabismus of the right eye, rigidity of the neck and slight retraction of the head. Motor paralysis of the left arm and leg with ankle clonus well marked on this side. He is very irritable and replies slowly when questioned. Temperature 100° F. to 102° F., pulse 45 to 56. It was suspected that another cranial abscess was forming. The mastoid wound was extended upward and forward for three-fourths of an inch above the temporal ridge, the dura was incised and a bistoury carried upward and inward into the brain substance until the abscess was reached. The sinus was also opened and a small elastic clot found near the jugular bulb.

From the time of opening the second abscess his recovery was speedy. *Campbell.*

A Case of Acute Internal Hydrocephalus Secondary to Streptococcal Infection of the Labyrinth.

Scott, London, Eng. (*Archives of Otology*, Vol. xxxvii, No. 2). A boy, aged 14, had a radical mastoid operation performed on account of an uncomplicated chronic suppuration of the left middle ear. There developed an acute internal hydrocephalus secondary to infection of the labyrinth and unattended by gross lesions such as brain abscess or meningitis.

Clinical tests for hearing were unreliable because the boy was mentally backward. Vomiting was the first untoward symptom; this occurred every day for five days after the operation. This was due, as autopsy revealed, to infection of the labyrinth. There must have been giddiness. Yet, of this, he made no complaint.

Severe headache masked all symptoms and it was produced by the increasing distention of the ventricles. Had the labyrinth been drained and at the same time the distended ventricles been relieved by lumbar puncture or puncture of the brain, life might have been saved. *Campbell.*

Report of a Case of Meningitis of Otitic Origin; Operation; Recovery.

KERRISON, New York (*Archives of Otology*, Vol. xxxvii, No. 2). A man, aged 34, with a fetid otitis media suppurativa which had persisted since childhood suddenly developed a severe left earache accompanied by high fever, severe occipital pains and vomiting. His mental condition was dull, leucocyte count 22,800 and polymorpho-nucleophiles 88 per cent. Unconsciousness developed, with contracted pupils and rigidity of the muscles of the back of the neck.

A mastoid operation was done and the incision carried upward so as to expose the middle fossa and temporo-sphenoidal lobe. The dura bulged, was congested and it was incised. Considerable oozing of cerebral fluid followed. A grooved director was passed in different directions into the brain substance. After being passed backward and inward for about two and three-fourths inches, there followed a free flow of cerebral fluid. The lateral ventricle had been entered. This completed the operation and gauze drainage kept the operative wound open for six weeks, while the cerebro-spinal fluid continued to flow. For the first 24 hours morphin was needed to allay the pain and after this the course was favorable.

Campbell.

The "Piano-String" Theory of the Basilar Membrane.

BRYANT, New York (*Archives of Otology*, Vol. xxxvii, No. 2). The author's conclusions are:

1. The basilar membrane is not essential to the organ of Corti, and when present is not furnished with the requisite length and mass of fibres to vibrate in sympathy with every note, even if the rest of the structures would allow it.

2. The basilar membrane is devoid of the requirements of a resonating body.

3. Helmholtz's "piano-string" theory of musical perception is without foundation in every particular. *Campbell.*

Sarcoma of the Middle Ear.

ZEBROWSKI, Warsaw (*Archives of Otology*, Vol. xxxvii, No. 2). The number of cases recorded is less than fifty. The author's is that of a man, aged 25, who has had a tumor growth in and about the left ear for eight months. There was total paralysis of the left facial nerve.

On operation the tumor mass was found to occupy the mid-

dle ear including the antrum and mastoid cells and also a space extending from the mastoid tip to the transverse process of the atlas. These soft gray masses were easily removed by a sharp spoon, but the growth promptly recurred and one week later a tumor the size of a pigeon's egg was removed from the left shin bone which on microscopic examination proved to be a round cell sarcoma.

On postmortem examination it was found that the growth filled the nasopharynx, had in part destroyed the sphenoid bone and joints of the occipital bone. The odontoid process had entered the skull cavity and destroyed the medulla. There were metastases in the lungs and heart muscle. The pericardium contained about 1000 cc. of reddish muddy fluid. The labyrinth was uninvolved. An interesting point was noted in this case as regards facial palsy in the differential diagnosis between primary and secondary sarcoma of the middle ear. This paralysis occurs only in cases of primary middle-ear sarcoma. *Campbell.*

A Case of Large Cholesteatoma of the Middle Ear and the Posterior Cranial Fossa, Cured by Radical Operation. No Recurrence After Eight and One-half Years.

ZIMMERMANN, Milwaukee (*Archives of Otology,* Vol. xxxvii, No. 2). A man, aged 36, in whose left ear polypoid masses projected from the external meatus. The middle ear was extensively necrosed. From the right ear a polyp and fetid cholesteatoma were removed. Eighteen months later an abscess formed and was opened on the right side of the neck one and one-half inches below the mastoid tip. This abscess cavity ran down along the sterno-cleido-mastoid muscle (Bezold's mastoiditis). The right facial nerve was paralyzed, there was a fetid otorrhea, the posterior-superior canal bulged and cholesteatomatous masses were removed from the attic. A radical mastoid operation was performed; when the cortex was removed a large cavity was found filled with a fetid cholesteatoma and when this was removed the lateral sinus and thickened dura was exposed. The medial portion of the posterior meatal wall formed a loose sequester.

Recovery was perfect and the facial paralysis partially cured. The eye can be closed completely but the angle of the mouth droops slightly. A permanent post-auricular opening was left. *Campbell.*

II—NOSE.

Nasal Headache as a Result of Hyperemia of the Central Nervous Organs.

RETHI (*Mediz. Klinik.*, No. 16, 1908) is doubtful of such a hyperemia as a general rule. He believes, however, that certain high degrees of nasal hypertrophy may cause severe headaches, but also says that the disappearance of the headache, after the application of cocain in the nose, is not caused by the astringent action, which theoretically would cause its increase, but due only to its local anesthetic action. *Horn.*

The Connection Between Diseases of the Stomach and the Nose.

HECHT (*Münch. med. Wochenschr.*, No. 12, 1908) shows by quoting two cases of empyema of the antrum of Highmore, how a continuous swallowing of pus, year after year, may set up a stomach catarrh of severe character, with loss of flesh, nausea and vomiting. All treatment by internists was without avail until a specialist discovered the source of trouble. The antrum was washed out and an amelioration of the stomach symptoms immediately followed. He deprecates the fact that so little weight is laid upon this connection in text books of diseases of the stomach. *Horn.*

The Treatment of Ozena by Means of Gargles.

SCHMIDT (*Münch. med. Wochenschr.*, No. 23, 1908) recommends gargling with closed mouth in cases where the crusts are impacted in the nasopharynx, and can not be loosened by nasal irrigation. A part of the water is forced behind the soft palate and the crusts are thereby loosened.

Horn.

The Nasal Reflexes.

KOBLANCK (*Deutch. med. Wochenschr.*, No. 24, 1908). In a most original and stimulating article, covered in the above heading, Koblanck quotes some remarkable cases, where a simple caustic of the inferior turbinate or the application of cocain to the nasal mucous membrane, has caused a marked improvement of various disorders connected with the sexual organs. During childbirth, the application of a 20 per cent cocain solution has often favorably influenced the pains and increased the strength of the uterine contractions. In dys-

menorrhea he considers it a most valuable diagnostic and therapeutic help. In twelve women who had always been sterile and where no cause could be found, a nasal treatment resulted in 9 per cent of them becoming pregnant, all within a period of a few months! In perityphlitis falsa he found it invaluable in differential diagnosis. In two cases of sciatica he secured a complete cure. In several cases of sexual hyperesthesia a cure also resulted as a result of local caustic operation. The results are not to be explained by suggestion and the various sudden deaths which have followed simple nasal operations are not due to the chloroform or cocain but to this intimate relation between the intumescent body and the central nervous system. *Horn.*

Nasal Dysmenorrhea.

KUTTNER (*Deut. med. Wochenschr.*, No. 24, 1908). Almost in direct opposition to the findings of Koblanck, contained in the article just reviewed, Kuttner says that "the center (standing in direct relation with the genital apparatus of woman) does not and could not exist." Although he has seen many cases of dysmenorrhea favorably influenced and even cured by local nasal applications he places it largely to the credit of suggestion, the general influence of the cocain, or the general well-being after the nasal trouble is corrected. Literature accompanies the article. *Horn.*

III—LARYNX.

The Pathology of Laryngocele.

SCHEVEN, Frankfurt (*Münch. med. Wochenschr.*, No. 9, 1908), recites one new case and reviews literature. *Horn.*

Clinical Notes on Laryngeal Tuberculosis.

BEVERLEY ROBINSON (*Am. Jour. Med. Sci.*, August, 1908). In the way of protective and curative treatment, two things are necessary: (1) rest for the larynx, and (2) inhalations. Personal experiences since 1885 has proved to the author the great value of the frequent, persistent, long-continued use of the perforated zinc inhaler with inhalations of creosote and alcohol, or, when there is much irritative cough, of beechwood creosote, alcohol, and spirit of chloroform in equal parts.

Richards.

IV—PHARYNX.

A Hairy Nasopharyngeal Polyp of Congenital Origin.

LEVINGER (*Münch. med. Wochenschr.*, No. 19, 1908) describes a case occurring in a 6½ months old female child. The tumor was pedunculated, 6 cm. long and 1½ cm. wide. It was covered with a delicate hairy epidermis. Through its middle was found a plate of cartilage. The growth was easily removed with the cold snare. Twenty-five case are known in the literature. *Horn.*

A Contribution to the Therapy of Angina.

BERLINER (*Deutsch med. Wochenschr.*, No. 13, 1908), uses a protargol salve combined with menthol, a small portion of which is introduced into the nasal aperture several times a day and appears to favorably affect the course of catarrhal and parenchymatous form. No cases are quoted. *Horn.*

The Local Treatment of Acute Inflammations of the Throat from the Standpoint of Pathology.

J. L. GOODALE, Boston (*The Boston Med. and Surg. Jour.*, June 25, 1908), has made a number of careful clinical examinations, both before, during and after treatment of acute inflammations of the throat, to see what the effect of treatment would be, local antiseptics and silver salts being used. As a result of his observation, he comes to the following conclusions:

1. "In a beginning tonsillitis antiseptic applications may perhaps be used with benefit and their effect, if any, will be to abort the local infection. If the disease is not checked at the outset by the sterilization of the parts, but if it proceeds to the formation of white spots in the crypts with systemic involvement, further application of antiseptics may not only be useless but harmful.

"It would appear possible that antiseptics may retard convalescence in two ways: First, by diminishing the number of bacteria in the crypts which are generating toxin and consequently prolonging the period required for the formation of the requisite amount of antibodies; second, by their destructive action upon the tissue cells and phagocytic leucocytes of the host.

2. "Forcible application of antiseptics may be followed by increased fever and cervical adenitis, indicating heightened

absorption of toxin into the system. The phenomenon may be compared to the depression which follows the injection of too large a dose of vaccine in cases undergoing opsonic treatment.

3. "In certain cases where acute tonsillitis appears to be aborted by local antiseptics, inflammatory manifestations may follow after a day or two in the neighboring regions and last for a number of days or several weeks. Here the possibility is suggested that the checking of the tonsillitis checked also the establishment of immunity, and that for its final accomplishment a longer period of growth of the organism upon the membranes of the nose, larynx, trachea or bronchi was necessary."

"If the conclusions to which I have come prove, on fuller investigation, definitely established, we shall have to modify our time-honored treatment of these affections. If I may venture to prophesy, our procedure will be approximately as follows: Active early local treatment as at present with guaiacum, silver preparations, etc., the avoidance of antiseptics when once the disease is definitely under way and, above all, complete local and general rest. It may be said with truth that the most vigorous and active measure that we can adopt in acute infections of the throat is a consignment of the patient to his bed, with avoidance of meddlesome interference."

Richards.

V—MISCELLANEOUS.

The Connection Between Disturbances of the Upper Air Passages and Diseases of the Genito-Urinary System.

SENDZIAK (*Mediz. Klinik*, No. 9, 1908) points out that diseases of the kidneys more often show themselves in the mucous membranes of the mouth and pharynx, than in the nose. We must be particularly careful, when a local cause is lacking, to regard with suspicion pallor of the membranes and unexplained hemorrhages, and examine the urine at once. In females, nasal disturbances of a genital origin are far more common; bleedings, hyperemia, secretion are commonly seen.

Horn.

Sodium Perborate.

HEYDENRICH (*Deutsch. med. Wochenschr.*, No. 3, 1908) recommends its use in ear discharges and as a post-operative

powder in nasal work. He claims for it a certain hemostatic action. *Horn.*

HARTMANN (*Deutsch. med. Wochenschr.*, No. 7, 1908) warmly recommends its use as a dusting powder in internal diseases of the larynx. *Horn.*

A Pin in the Left Bronchus.

V. SCHROETTER (*Deut. med. Wochenschr.*, No. 15, 1908) gives an interesting case of a 12-year-old girl who aspirated a shawl pin 74 mm. long with a glass head 8 mm. in diameter. The pin was embedded for twenty-one days and removed by a direct bronchoscopy, through a tube 21 cm. long and 8.5 mm. in diameter, at the first trial. No reaction; complete recovery. *Horn.*

A Universal Demonstration Lamp for Laryngoscopy, Tracheaoscopy and Esophagoscopy.

HORN, Bonn (*Deut. med. Wochenschr.*, No. 17, 1908). The handle was designed for use with the Kirstein electrical headlight. By a simple vertically placed joint, the lamp can be swung out the line of the tube and the mandrin removed, a cocain application made, or the coughing of pus against the mirror prevented. It was designed especially to replace the Casper electroscope, which because it employs a prism to cast the light at right angles down the tube occupies half the field of view and when once in position can not be removed for manipulative procedure, thus making it impossible for demonstrative work. *Horn.*

Bronchoscopy and Foreign Bodies.

VON EICKEN (*Deutsch. med. Wochenschr.*, No. 17, 1908) gives a short but very interesting review of the work of the Killian clinic for ten years, in the removal of foreign bodies from the bronchi. In fifteen cases, the foreign body was found twelve times. Twice it was impossible to certify the diagnosis and once the severe wound in the bronchus could be only explained as a trauma resulting from a foreign body. Seven cases were operated on with local anesthetics and eight in general narcosis. Of these eight cases four were operated on in America where the use of a general anesthetic is common and the other four were all children. The great advances which have been made are due largely to Killian's

assistant, Brünnings, whose telescope tubes and electrical operating lamp leave little to be desired. One fatal case only mars the statistics; a baby who had aspirated a large bean and was brought into the clinic three days after the accident with a severe pneumonia. *Horn.*

The Operative Procedure in Deep Lying Abscesses of the Tongue.

BRUNK (*Deutsch. med. Wochenschr.*, No. 23, 1908), recites two cases and gives literature. He claims that the opening of the abscess from the mouth is uncertain and dangerous. Recommends the external route through the musculus hypoglossus. *Horn.*

Lactic Acid Bacilli Cultures in Nose, Throat and Ear Work.

H. HOLBROOK CURTIS (*New York Med. Record,* July 11, 1908) has been experimenting with culture of Massol's bacillus, introduced directly into the nasal cavities in cases of empyema of the accessory cavities, and in atrophic rhinitis. A cubic centimeter of the culture broth is sprayed under pressure so as to reach the ethmoid cell orifices, the theory being that the bacilli will find their way into the ethmoid cells, and accessory cavities, and by their secretion destroy the pyogenic bacteria which are causing the disease. Five cases of sinus and ethmoid trouble, and two of atrophic rhinitis are cited, in all of which rapid improvement took place. The announcement is preliminary only. *Richards.*